THE DEGENERATIVE BACK

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THE DEGENERATIVE BACK

AND ITS DIFFERENTIAL DIAGNOSIS

by

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FOREWORD.

Many a medical practitioner must have been confronted at times with cases which failed to respond to the traditional methods of treatment and have felt a challenge to his professional curiosity and ingenuity. If, moreover, his practice was of a kind to bring to his consulting room considerable numbers of patients suffering from a particular ailment, only some of whom found relief from the normal treatment, he will have been stimulated to give special thought to the matter and to study his records. Details, general information and inferences, however, would serve little useful or durable purpose if they were not digested and presented in palatable form to the world. Indeed, the physician into whose lap such windfalls have dropped is under a moral obligation to share them, so that others, notably doctors and patients, may be allowed to benefit by what may prove to be valuable and helpful information.

Even during my training, I, personally, was struck by the unsatisfactory nature of our knowledge of the true character of all those forms and gradations of low back pain and sciatic symptoms which hamper so many people in their daily lives.

The recent development of neurosurgery, especially the surgical treatment of sufferers from the above complaints and symptoms, provided me with an opportunity, precisely in this medical specialty, of giving rein to my preoccupation with this group of patients. By degrees I became convinced that there were gaps in what had come to be the fixed ideas about a simple aetiology of these clinical pictures (including hernia nuclei pulposi and sciatic neuritis). The neurological symptomatology was apparently less watertight than had been supposed and the erratic results of surgical treatment, despite increasing technical perfection, continued to be puzzling.

The fact that spinal infirmity in all its manifestations can be regarded as a disease or ailment which, apart from its repercussions in the national economy, may be a decisive factor in individual self-realisation and happiness, tipped the balance in favour of my decision to explore precisely this field of knowledge.

Broadly speaking, the plan of this book reflects the history of my personal approach to this problem. The intention behind the detailed presentation of

the points of enquiry in Part I is to whet the reader's curiosity as to the unknown and the uncertain, matters which are discussed in Parts II and III in the light of the literature and of our own practical experience. I hope that my conclusions will be conducive to a better understanding of the diseased of weak back and of those sufferers who are burdened with it.

Wassenaar, March 1959 P. R. M. J. HANRAETS

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INTRODUCTION

Complaints of back trouble and radicular pains may well come within the province of the neurosurgeon's clinical and therapeutic work. The treatment of patients suffering from these complaints has made it clear that the way the human spine is constructed is liable to vary in different individuals, sometimes producing symptoms of disease. From the clinical reports studied and the vast revelant literature, we came to see that these symptoms seldom result from a single pain-provoking mechanism. It also became apparent that it is not enough to attack the most patently disturbing factor, but that the secondary circumstances partly responsible for the condition of the patient should likewise receive attention. In the light of our experience, we came to the conclusion that hernia nuclei pulposi should by no means be considered as the only condition of low back pain and radicular symptoms that calls for surgical intervention. Finally, the syndromes often thought to point to the existence of hernia nuclei pulposi were certainly not, we found, pathognomonic for this affliction.

The results of our studies, pursued on these premisses, are the subject of this book.

Although the words "disc" and "hernia" may occur more frequently than in many an article on the matter, it has not been our aim primarily to describe hernia nuclei pulposi and its pathology; we are concerned, rather, with the complex problem in which the complicated pathology of this syndrome is imbedded and with the constitutionally weak back in its manifold manifestations. In the chapter on the symptomatology of the degenerative back, the symptoms are compared with those brought about by hernia nuclei pulposi. We have not merely enumerated them, but have tried to account for the reason and cause of their occurrence.

Our opinion that there can be a constitutional predisposition to lumbar complaints is borne out by the fact that a morphologically demonstrable abnormality will distress one patient and not another, whereas similar distress and symptoms may well occur without any such abnormality; likewise by the proved

idism. On the other hand, the indicated course may be to leave the patient to grapple with his aches and pains, rather than to run the risk of upsetting his already labile psychic balance as the result of an operation which, on account of his mental and physical condition, stands no chance of producing satisfactory results, whatever the cause of the complaints may be. To appraise the therapeutic possibilities, it is necessary to be able to evaluate those manifestations of the constitution which we call a weak, degenerative back, a reality which is recognised in popular parlance, but in medical circles is often ousted by more resounding diagnoses, such as neuritis and hernia.

Recognition of the signs of an endogenously weak back will undoubtedly be weighed in the balance when we are consulted as to vocational fitness, choice of work and the connection between trauma and back complaints.

There is a vast literature on spinal abnormalities. We felt that the results of our study of this literature should be recorded *in extenso*, in order to provide the reader with a full survey of the most important reports issued up to 1958. In claiming that it is comprehensive, we mean that it is so only in regard to the variety of the subject, not to the number of authors who have dealt with it. There are a great many reports, valuable in themselves, which approach the subject from the same angle. For reasons of space, we have selected from these the pronouncement of one author only, namely whoever was first in the field or specially commendable for his clarity. This accounts for the absence of the names of numerous authors, equally well known, and mention of their valuable contribution.

Case histories form a useful link between the opinion of the author and the conviction of the reader. The many variations in the structure of the human back are associated with a large number of case histories, each of which would be worthy of note. After sifting them, we nevertheless decided to mention only a single detailed example, not wishing to interrupt fluency through the constant interpolation of examples. Some special cases are presented schematically in the last table of the chapter on Treatment.

The present study has been confined to complaints of low back pain and the affections of the lowest extremities which often accompany it. Where relevant, we have touched on, but not described in detail, those back troubles which are due to exogenous causes (traumata, infections and so forth) and the complaints and symptoms of known and described syndromes (such as the whole gamut of rheumatic syndromes).

The data presented in this book were collected jointly by a team consisting of neurosurgeons, psychiatrists, neurologists and a radiologist and, where appropriate, in collaboration with an orthopaedist, general surgeon and pathologist.

existence of numerous variations and anomalies in the constituent parts of the spine. In another chapter we shall explain what is meant by "constitution".

Once it has been fully recognised that a given tendency may predispose the patient to lumbar trouble, it will be evident that it is necessary to establish the extent to which this tendency constitutes an aspect of the patient's structure *in toto*. It is with this in mind that we have given ample thought to the question as to whether a degenerative psyche is a concomitant of the degenerative back. Finding that this was so in fact, we next asked ourselves whether the signs of constitutional psychic lability manifested side by side with those of an endogenously degenerative back have a reciprocal influence upon each other and, if so, how. It ultimately transpired from our study of the constitution of some of these cases that the constitution is an essential factor in the pathogenesis of spinal complaints. Here we should point out that an endogenously labile structure of the personality, which implies abnormal psychomotor behaviour exhibited by abnormal motor activities, is capable of weakening what was congenitally and initially a normal back, leading to a pathological condition.

In many cases the connate factors predisposing the subject to pathological symptoms have proved to be clearly demonstrable. We thus came to regard a personality structure of this kind as expressive of degeneration and collected its manifestations in the human back under the term "The Degenerative Back".

We hope that a patient will never be told, or at most in exceptional cases, that he or she has a "degenerative back". This diagnosis is the physician's affair and should not come to the ears of the patient. Many people with back complaints and radiating pains believe that they are suffering from a "hernia"; and, in fact, this often is so. If they are mistaken in this belief, it may be wiser not to disabuse their minds, for it is precisely patients with a labile psychic make-up, which is often a concomitant of the degenerative back, who find relief in the diagnosis of the now popular "slipped disc" as something to hold on to. But the medical practitioner should be able to distinguish between certain modalities of a back complaint and to treat it adequately with due regard to the well-being *in toto* of the patient under his care.

It should be realised, before all, that possibly every sufferer from back complaints needs some sort of therapy, but that not every patient is curable. Although pragmatism is to be deprecated, the practitioner may be failing in his duty if, by adopting too conservative an attitude, he deprives his patient of the chance of relief through surgical intervention, merely because he or she is not suffering from a hernia nuclei pulposi. What we treat the patient for is not the "hernia", but the distress impairing health or exacerbating inval-

PART I

POINTS OF ENQUIRY

STATEMENT OF THE PROBLEM

Although the book was actually written by a neurosurgeon, we do not think the point of view expounded has an obviously neurosurgical bias. In the bibliography, reference is made to specialists in virtually every branch of medicine. As we read some quotations, we may not be able to escape the impression that a particular author is expressing the specific views of, say, a neurosurgeon, a neurologist, an orthopaedist or a rheumatologist. The neural therapeutist and chiropodist likewise voice their opinions and, although we do not read much about them, the family doctor also, naturally enough, has his own ideas about the treatment of a painful back. And it is well that it should be so, particularly as all these practitioners receive the patient for treatment, often at different stages of the illness, and each deals with an individual facet of the complicated syndrome.

The progressive differentiation in the technique of medical examination requires co-operation in a number of spheres. True, a man working alone can, with knowledge borrowed from elsewhere, score successes in practice; but great advances and a clearer grasp of the problems can be obtained by free exchange of experiences between specialists. The more team-work there is, the more intensive and extensive will this exchange be, until – and that time *is fast approaching – there is no room for a narrowly circumscribed specialist* approach to a particular problem. This most certainly applies to spinal diseases. It would be hard to single out any province of medicine in which the practitioner is not confronted with the problems surrounding the diseased back. For that reason this book is addressed to the family doctor, the specialist applying conservative treatment and the surgeon. It is the author's fervent hope that it may add its quota to any synthesis that may eventually be arrived at, capable of coping with the question of the most effective treatment for a patient suffering from a pathological back.

CHAPTER I

STATEMENT OF THE PROBLEM

The problem of low back pain and the radicular syndrome as it stands today

Mention of back complaints and the kind of pain usually associated with sciatica is to be found all down the ages from some of the most ancient documents extant.

Our direct ancestors complained of a "crick in the back"; Couperus * and other novelists of the same period drew a vivid picture of the *weak back* when presenting their leading female characters. Rising neurologists diagnosed it as *sciatic neuritis*; in the pre-neurosurgical period the problem was attacked from the *orthopaedic* point of view. Then came a time when a *hernia nuclei pulposi* was thought to be responsible for all, or nearly all, low back pain and radicular pain; and there is reason to believe that the indication for neurosurgical intervention in suspected cases of a "slipped disc" was accepted with rather too much alacrity. Nor were the results of that procedure highly satisfactory: there were many cases of recurrency and negative explorations were too common. The psychic aspects of the clinical picture were either neglected or underestimated, especially in the period just referred to.

As a natural reaction to this state of affairs, the profession tended to become chary of admitting an indication for surgical therapy; indeed, some of the more conservative members declared that "hernia nuclei pulposi is no longer treated by operation". There was a reversion to the usual conservative treatment for crick, lumbago and sciatica which had been applied before hernia nuclei pulposi became known as a defined disease.

Rest and warmth were known by experience to alleviate pain and discomfort in the back and legs. The advice given varied, according to the views of the examiner and therapeutist, from complete rest in bed on a rigid support to a plaster bed, lumbar corset, manipulation under an anaesthetic, formic acid, irgapyrine and euphylline injections and the application of salicylic acid preparations Treatment by radiation, Novocaine injections in the hiatus

* A well-known Dutch novelist, born in 1863, belonging to the literary group known as the "Tachtigers" – *i.e.*, men of the eighties.

cured patients had not been suffering from a hernia nuclei pulposi at all. This is tantamount to saying that the strongest argument in favour of the conservative treatment of hernia nuclei pulposi is the success of this form of therapy. But the argument falls to the ground if it is not an established fact that the patients were suffering from hernia nuclei pulposi; and this was precisely Krischek's point. In that case it was merely symptomatic treatment for back and radicular pain from an unknown cause.

Pondering these and similar statements, we asked ourselves over and over again, as will be apperent in the following pages, what these (unknown) disorders could be that so deceptively simulated the symptoms of hernia nuclei pulposi.

Moreover, in view of the not always satisfactory results of surgical therapy, some neurologists and neurosurgeons began to question the authenticity of the diagnosis in the first place.

Improved diagnostics. The method of diagnosis was improved, the cases were selected with greater discrimination and the indications were better defined, a state of affairs assisted by the fact that many patients with complaints pointing to hernia underwent extensive conservative treatment before surgical intervention was hinted at.

Recurrency among genuine hernia nuclei pulposi patients. At the same time articles were published, both at home and abroad, in which the possible causes of the failure of surgical treatment were discussed. And, although the findings at re-operation did not always provide enlightenment, suggestions as to possible causes led gradually to improved technique and this in turn eventually produced better results.

This notwithstanding, the explanations offered were not always watertight. Why, for instance, should root adhesion occur in one case after a laminectomy and not in another? Why did the extirpation of a particularly large hernia nuclei pulposi often leave the patient in more discomfort than that of a small one?

Negative exploration for hernia nuclei pulposi. Nor had any satisfactory explanation been forthcoming for the fact that, upon exploration, nothing abnormal had been found in the discs of patients suffering from the *typical* low back pain and radicular pain commonly associated with a hernia nuclei pulposi, although thorough examination by every possible means had ruled out the existence of all other pathological conditions.

This incongruity led several investigators at home and abroad to assume that the vertebral column and parts closely adjacent to the cauda equina must be subject to *other organic disorders* liable to complicate or simulate the complex

sacralis, neural therapy, treatment of the tonsils with cocaine, injections in the ganglion spheno-pallativum.... all these were tried out. Others again resorted to a more active form of therapy, prescribing certain gymnastic exercises and massage, with varying success. The dominating idea has often been that every conservative treatment should be given a chance first, regardless of the cost and time entailed. A critical study of these methods has shown them often to be treatments for symptoms of unverified lesions. Some patients may be completely cured of an attack of uncomplicated sciatica by a period of rest in bed, just as others with a strained muscle may find that work, massage and physical exercises relieve them of their pain.

A simple chart published by Junkersdorf puts this matter into clear perspective, *viz.*:

46 *unverified* cases of "hernia nuclei pulposi".

After 9 to 12 weeks of conservative therapy:		
	Symptoms cleared up	51%
	Improvement	37%
	Patients "recovered"	88%
After one year:	Notwithstanding avoidance of hard work, notwithstanding change of work, notwithstanding repetition of treatment,	
	Recurrency	15%

Junkersdorf declares that the treatment he advised cured or greatly improved a large percentage of his patients "*suffering from a hernia nuclei pulposi*", yet he clearly implies in the same article that the diagnosis was not definitely established; nor had it been verified by myelography.

Krischek describes 150 *unverified* cases, likewise lacking myelographical corroboration:

	150	
Symptoms cleared	52	
Greatly improved	59	
		74%
Little improvement	27	
No change	12	
		26%

Considering how often erroneous diagnoses are still made, despite careful neurological examination in a competent clinic, X-ray exposures and myelography and data on lumbar puncture and cerebrospinal fluid, also considering how often the expected hernia nuclei pulposi was not found at operation, it is fairly safe to assume that an appreciable percentage of these entirely or partly

CHAPTER 2

DEVELOPMENT OF METHOD OF OPERATION

The experience that gave rise to the development of a modified method of operation and to revised views as to the causes of low back trouble and radicular pain

Those who are constantly engaged in the operation theatre on the laborious routine manipulations involved in the opening and closing of surgical wounds have ample opportunity for reflection upon the various problems associated with the operation.

The adoption of a different approach is by no means an abrupt decision on a particular day to depart from the familiar procedure; it is, rather, a slow gestation, the gradual unfolding of an idea which is tried out again and again at appropriate operations where former and present experiences are correlated and re-interpreted in the light of that idea.

The operating method about to be described can be briefly summarised as a more lateral exploration of the roots in their course down to the foramina intervertebralia.

It will be shown in the ensuing pages how, exploring for a disc lesion, our tracking of the course of the roots became more and more lateral in our quest for solutions to unresolved problems.

RESULTS OF THE "NEGATIVE EXPLORATIONS"

Upwards of 3000 laminectomies were performed in the St. Ursula Clinic at Wassenaar for the relief of low back pain and radicular pain. More than 2000 cases altogether, comprising half the above laminectomies plus 500 cases elsewhere, provided the surgical experience of the author himself in this particular field.

From this material we have collected the cases in which *no hernia nuclei pulposi* was found at operation, in spite of the fact that the clinical picture was indicative of that condition and that myelographic examination pointed more or less definitely to its existence.

We made a point of following the post-operative course and found that improvement and even complete recovery occurred in a number of these cases, *viz.*,

of complaints and symptoms of hernia nuclei pulposi. The main object of the present study is to muster evidence of other organic disorders affecting the supporting frame and root system.

After this survey, we can more emphatically refute the contention of some medical practitioners that it would be better if fewer operations were performed for hernia nuclei pulposi. This, we think, is a mistaken attitude, for there is practical proof, not only that there is an ever-increasing number of well-diagnosed cases admitted for operation, but that recent results have fully vindicated the decision. Furthermore, the latest developments in neurological and radiological examination and the better-informed interpretation of abnormalities found at operation go to show that a new field is opening up, in addition to the indication for hernia nuclei pulposi, in which surgical therapy can alleviate and, indeed, relieve patients of their complaints.

substantial difference. Besides, the roots in the dural sac pass the level of the decompression in their course to lower innervation regions than the site of the exploration. *E.g.*, exploration takes place at L.4-L.5 if a radicular abnormality of the L.5 nerve is inferred from the clinical-neurological picture; but at this level the intradural roots proceed to the S.1, S.2, etc. innervation regions. If the syndrome or pain clears up in the innervation region of this L.5 root after the operation, the obvious explanation would seem to be that the decompressive effect of an explorative, so-called "negative" laminectomy was upon the *root* in that particular area and not upon the whole dural sac. Although we surmised that it was the roots that were decompressed in some negative laminectomy cases, we did not yet know, having ruled out a hernia, what had brought the roots to this pass.

It seemed to us that there must be other causal organic conditions, besides hernia nuclei pulposi, which simulate the radicular syndrome produced by hernia nuclei pulposi; and, in view of the relief derived from a *decompressive* intervention, we argued that these complaints might well be brought about by *compression of the root.*

Results of Rhizotomies

From the large number of cases treated by rhizotomy to relieve radicular pain, however caused, we selected that group in which exertions raising the spinal fluid pressure, such as coughing, sneezing, straining, symptom of Naffzinger, etc., brought on or exacerbated the pain. It transpired subsequently that in very many cases the pain was relieved to a great extent as a result of the operation. This suggests the following argument: The reaction to jugular pressure shows that the resulting intracranial rise in pressure is propagated via the cerebrospinal fluid spaces; with monoradicular pain, any access of pressure in this whole system (veins, intracranial fluid spaces, dural sac) manifests itself as a sensation of pain in the innervation area of one root. It may be supposed that the pain irritant set up by coughing, sneezing and so forth does not arise locally through swelling of the veins, but that the pressure in the fluid spaces is heightened as the result of this venous blood pressure. Further protrusion of a hernia nuclei pulposi is sometimes said to be responsible for the onset of monoradicular pain under these circumstances; but the symptom also occurs in similar cases in which no hernia was found at operation. After (selective) sensory rhizotomy, the monoradicular syndrome cleared up in these "negative" cases as well.

The group of rhizotomies involving the above combination of phenomena

Recovered	53	(40.1%)
Improved	30	(22.7%)
Unchanged	49	(37.1%)
	132	

Several conclusions presented themselves:

1. It can be inferred from this material, in passing, that the value of myelographic examination is comparatively limited.
2. We had to ask ourselves whether, in the cases of post-operative recovery, the organic character of the complaints had been correctly established pre-operatively.

We shall not deny that, where there is some degree of psychically determined functional superposition of the complaints, an explorative operation may act upon the patient so potently as to suggest to him that he is cured, temporarily at any rate. This may admittedly happen, but it is improbable that it did to *all* the patients who were considered before the operation to be suffering from complaints of organic origin. Moreover, it is a recognised fact (Zukschwerdt and Krischek) that some patients, declared by the psychiatrists to be psychically normal, present to the neurologist the typical clinical picture of a hernia nuclei pulposi, yet are completely cured of their symptoms after an explorative laminectomy with negative findings (see also Krischek's statistics, p. 67).

3. How then did such laminectomies come to have this favourable effect precisely in cases where suggestion was thought to be an improbable factor? If, as under 2, we cannot believe that *all* the patients who responded to the treatment were psychically unbalanced, we are led to assume that the mechanism of the operation itself was responsible for the disappearance of the symptoms. It is difficult to see how the effect of this mechanism can be anything other than decompressive, seeing that at least one arch is removed and the ligamentum flavum extirpated, with the result that the dural sac is able to bulge somewhat in a dorsal direction. When this takes place, the roots are pulled a little farther out of their foramina. Possibly, too, after removal of the parts lying dorsally over the dural sac, the latter and the roots are better able to give way (in a dorsal direction) to a ventral prominence. No manipulations other than those described above take place during an explorative laminectomy, the decompressive effect of which is generally recognised as being plausible.
4. Can we now suggest or demonstrate a specific reason for the cure or improvement by decompression alone in the cases we are considering?

The dural sac itself is a comparatively flabby bag in which the roots run freely; hence a local decompression of the dural sac is unlikely to make any

List of Causes of Low Back and Radicular Pain

Reasoning from the provisional conclusions drawn from the foregoing, our next postulate was that other disorders besides hernia nuclei pulposi must affect the lateral two-thirds of the root and ganglion.

In order to discover what these disorders could be, we drew up a list of the various pathological conditions liable to produce low back and/or radicular pain so as to be able to differentiate further from this list.

1. Traumata	Fracture of the vertebral arch, vertebral body, spinous process; traumatic spondylolisthesis; haematoma in muscle, joint or vertebral canal.
2. Anomalies	Spina bifida occulta; stenosis of the vertebral canal; spondylolysis and spondylolisthesis; transitional vertebrae, fused, fractured and wedged vertebrae.
3. Disorders of the:	
muscles	Lumbago, muscle rupture.
ligaments	Hypertrophy of the ligamentum flavum; asthenia.
intervertebral discs	various forms of ailment from intervertebral discs;
joints	gout, rheumatoid arthritis, arthrosis deformans, Bechterew's disease.
4. Changes in the vertebral column due to static factors	Scoliosis, kyphosis, lordosis, etc.
5. Neural disorders	All disorders of the central nervous system; arachnitis spinalis; radiculitis, neuritis, polyneuritis.
6. General surgical and gynaecological disorders in the abdomen	Cold abscess, chronic appendicitis, colitis; stone in the ureter, pyelonephrosis, anomalies of the ureter; affections of the female organs, such as changes in posture, tumours and chronic inflammations, prolapsus vaginae, pregnancy.
7. Toxaemia	*E.g.*, lead poisoning.
8. Inflammations	Influenza, smallpox, typhoid fever, scarlet fever, diphtheria; sepsis, osteomyelitis, arachnoiditis; syphilis, tuberculosis.
9 Tumours	Sarcoma, carcinoma, haemangioma; neurofibroma, meningioma, etc.
10. Reticuloses	Lymphadenoma, Gaucher's disease, etc.
11. Diseases of the blood	Leukaemia, Kahler's disease, Werlhof's disease, etc.
12 Vascular diseases	Vascular spasm, embolism, thrombosis; arteriosclerosis, aneurysm, varices; haemangioma, angioma.
13. Skeletal diseases	Von Recklinghausen's disease, Scheuermann's disease, Paget's disease, etc.; Albright's syndrome; osteomalacia, osteoporosis (pre-)senilis; all forms of symptomatic osteoporosis.

provides the material basis for the following reasoning: A direct pain reaction of a root due to a sudden rise in pressure in the lumbar sac must inevitably be induced in that part of the dura and root system which stands in open communication with the cerebrospinal fluid spaces. The arachnoid invaginates to the extent of some millimetres. The abrupt rise in fluid pressure will act as an irritant upon parts of the root adjacent to this invagination, directly if the pressure is transmitted, or indirectly if it causes slight displacement of the root and ganglion.

It is generally agreed that the influence of increased pressure in the dural space is confined to the interior of the intervertebral foramen, *i.e.*, to the dural space, nerve root and ganglion.

Aetiological indications were found in the extrathecal course of the root, such as "radiculitis" and hernia nuclei pulposi, in a great many patients complaining of the onset or exacerbation of pain under the pressure-raising stresses referred to.

In view of what has been stated, however, it would be reasonable to assume that the aetiological origin of radicular pain would attack the part of the root lying centrally to the intervertebral foramen, or at most within that foramen. Because it had been found that radicular pain could be relieved by cutting the sensory root, this was a sufficient argument in our working hypothesis to warrant the assumption that the aetiological site of this pain must be peripheral to the site of the section. (For counter-arguments and contradictory views see Part III, page 359.) The most common and oldest technique is to cut the root intradurally. For reasons to be explained, we have adopted as our routine method since 1949 *extradural* rhizotomy in the root sheath. The sensory root is then cut in the root sheath at 4 to 5 mm from the dural space, *i.e.*, exactly peripherally to the invagination of the arachnoid.

As an enquiry has shown, the results of sensory rhizotomy in the root sheath are more satisfactory than those of the intradural method (see page 635).

To sum up the foregoing we can say that increased intradural pressure was considered to cause or aggravate monoradicular pain by irritating the intrathecal or extrathecal root, but centrally to the intervertebral foramen. The remission of this monoradicular pain after sensory rhizotomy led us to attribute the cause of this pain to the course of the root peripherally to the site of section. Combining the two considerations, we felt there was good reason to locate the *aetiology of the radicular pain* as central to the intervertebral foramen and peripheral to the invagination of the arachnoid into the root. This is virtually the *lateral two-thirds of the root plus the ganglion.*

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14 Deficiency diseases	Avitaminosis B1, avitaminosis D, *e.g.*, rickets; calcium and protein deficiencies, *e.g.*, osteomalacia.
15. Parasitic infestations	Echinococcus in vertebral canal and muscles.

We asked ourselves whether it was right, in this quest for hitherto unknown disorders, always to think of low back pain *and* radicular pain as necessarily associated symptoms. These two pathological conditions are so often combined and so frequently merge one into the other that they would obviously seem to originate from a common cause. So far, we have been guided by the picture of radicular pain and our search has brought us to the lateral two-thirds of the root and to the ganglion; this corresponds spatially to the lateral part of the bony spinal canal and the intervertebral foramen. Evidently, therefore, we should pursue the quest among those pathological conditions which produce radicular pain and in which, at the same time, patients may suffer from low back pain; and, besides the clinical pictures already known, we should for the time being have to eliminate those which, as far as we were aware, gave rise only to low back pain.

All the patients from whom our records were taken were observed by neurologists, psychiatrists, radiologists and resident physicians in the St. Ursula Clinic. Complete blood, urine and spinal fluid tests were invariably made and, where necessary, special laboratory tests (calcium balance, test for endocrine disturbances and so forth). We verified that all this really had been done in the clinical histories relating to the group of negative explorations in which we were so particularly interested

The vast majority of the patients admitted to the St. Ursula Clinic, who were stated, erroneously, to be suffering from a "slipped disc" or a vaguely defined syndrome in the lower part of the back or the legs, were diagnosed by us predominantly as cases of arthrosis deformans, tumour, tuberculosis, polyneuritis, serious central nervous or psychic disorders It would be unreasonable to suppose that a patient who had undergone a searching examination by a team of specialists as above could be suspected of suffering from any of the ailments included in our list if there was nothing to show that he was. The early symptoms of Kahler's or Bechterew's disease or of haemangioma might possibly have been overlooked, or perhaps the first signs of what was later to be manifested as a malignant tumour; but the number would be negligible among the large group of roughly 3000 persons examined.

We were therefore justified in assuming that sufferers from pain in the back and legs, in whom no hernia had been found at exploration, with some exceptions, were likewise not suffering from any of the known ailments included in our list. The cause of these complaints *must* therefore be something which we at operation "could not see or detect, or else something very unusual". Now, what we do not see at the operation is the psyche and the

functioning of the painful back and limbs; "something very unusual" is synonymous with something out of the ordinary, something to which not everyone is subject.

The logical inference from this was that we should seek for the causes of inexplicable low back pain and radicular pain in *psychical disorders* and *pathological movements of the supporting frame*, while the second assumption appeared to lead directly to *constitution* and *anomaly*.

Travelling along the root in search of causes for radicular pain, we had come to the intervertebral foramen, which is formed in part by two sections of the intervertebral joint. As long as the movements in the foramen take place physiologically, there is little reason to expect that they will produce pathological conditions associated with radicular pain. The question therefore arose as to whether radicular pain – and low back pain – is more common in people who move *other* than in the normal way.

Disorders Arising from Defects and Deformities of Vertebral Column and Extremities

The impractibility of making a quantitative comparison between the incidence of back complaints and radicular pain in individuals suffering from and not suffering from lesions or deformities at once became apparent. We were struck by the number of patients sent to the polyclinic, suspected, though not manifesting the symptoms, of a lumbar lesion, yet all presenting some deformity, in varying degree, of one of the lower extremities. *E.g.*, advanced arthrosis or ankylosis of the hip or knee joint, coxa vara, amputations, hammer toes, either operated upon or not; flat feet, various forms of talipes, painful residual conditions of thrombosis, varices, wounds. Many were lame and all, without exception, adopted a gait calculated to spare the affected limb. They had developed an abnormal, *i.e.*, a pathological motor system. Fifteen per cent. complained of pain in the region of the deformity, 60% of pain elsewhere in the deformed leg, 27% of pain in the contralateral extremity, 83% of pain in the back; 16% complained of exacerbation under pressure-raising strain; approximately the same percentage seemed to us to present the symptoms of nuclear retropulsion. Of a total of 50 individuals, six were admitted to hospital for observation; the myelograms of four showed filling defects and a hernia was removed from three of these patients by operation. The majority stated that their symptoms varied with the weather.

We learned from some patients that they had been lame for years without being aware of any pain. Among them, however, were some who came to

consult us about other complaints, such as headaches, fits, trouble in the arms, etc., and who only mentioned pain in the lumbar spine or one of the legs when questioned; they had thought that "that" was part and parcel of their particular disability and it had never entered their heads to seek medical advice for that alone.

Let one case serve to illustrate this. A man of 33 had an accident in which his right knee was jammed between a wall and a motor-car, as the result of which that joint became stiff. Upon recovery, he was able to walk without pain, though the knee was stiff and slightly flexed, he walked with a limp and always threw his weight on the stiff leg when turning. The spinal column and the other leg had not been examined radiologically after the accident because he had not complained about any other parts of the body. At the end of a year the patient became conscious of a dull ache in the low back, which grew worse in the ensuing six months; he was aware of slight pain radiating from the buttock and calf in the left leg right down to the little toe, especially when rising from a sitting posture and after exertion. Neither coughing, sneezing nor straining worsened this pain. Movement of the vertebral column was fairly flexible, without scoliosis or corkscrew phenomenon; Lasègue left weakly positive; reflexes and sensitivity normal. X-ray showed slight arthrosis around the vertebral joints L.V–S.I and in a minor degree at L.IV–L.V.

Roughly the same course of events and the same ultimate condition – though without radiological changes – were observed in the majority of the other patients. Over and over again we found that, after a transitional period of one or two years, during which the patient was free from pain, a syndrome gradually began to develop which we ascribed to excessive pressure bearing on the supporting frame. The clinical histories showed clearly that most of the patients concerned were young people with sufficient stamina to pursue a vigorous life, in spite of their disability, as though there were nothing wrong. This quiescent period of two years preceding the symptoms was exceeded more often in the cases of older patients leading a quieter life or of those who, having an indolent psyche, naturally went about everything at a slower pace.

These observations convinced us that the delayed complaints were secondary to the residual condition of the lesion so plainly apparent elsewhere in the body. To us it seemed that the only possible connection between the two must be the pathological movements to which the affected extremity and certain parts of the vertebral column were subjected. At first it was difficult to see why monoradicular pain should result. We could assume that a chronic habit of making pathological movements would put excessive strain upon the mobile parts of the vertebral column, or, more precisely, the disc and the vertebral joints. Actually, however, the patients did not move at all as though they had

a disc lesion and, in fact, retropulsion was discovered in only three of the fifty patients. Hence the obvious inference was that it must be the vertebral joints which underwent changes as the result of this chronic overloading; radiographs, moreover, showed the existence of spondylarthrosis in several patients.

Clearly, changes in the vertebral joints at the site of the intervertebral foramen would cause monoradicular disorders. (We should not forget that this foramen is formed by two mobile parts of the vertebral column, *i.e.*, the rims of the upper and lower intervertebral joint.)

Our argument now was that healthy individuals, who had never had these complaints before, behaved in a way which brought about pathological movements in the vertebral joints, hence in the foramina, after which they began to complain of pain in the back and/or in one leg.

It had already been our firm belief that syndromes of unknown aetiology were induced in many people by root *compression* and lack of adequate space around the lateral two-thirds of the root and the ganglion, that is to say at the entrance to the foramen. The evident assumption was, therefore, that these chronic pathological movements changed the foramina to such an extent that compression or shrinkage of space resulted. This was confirmed subsequently by other authors and by our own observations.

Complaints Associated with Neurological Disorders Accompanied by Paresis and Ataxia

If pathological movements due to manifest orthopaedic abnormalities were capable of bringing about changes in the intervertebral foramen, it was reasonable to suppose that pathological movements from other causes could have a like effect, such as *muscular disorganisation* (cold, lesion, sprain, contusion, etc.) involving a degree of contraction that forces the sufferer to assume an unnatural posture. The same applies to *neurological disorders* and, more particularly, neuritis which, through the pain it causes, is liable to set the same mechanism in motion. But these two examples, being so proximate to the area with which our argument is concerned, are perhaps too obvious; it might be countered that neuritis spreads over the root and by that alone produces pain, or that a lesion of the muscles in or near the back is felt as low back pain.

It would be far more to the point if we were able to show that, as the result of pathological movements induced by other neurological disorders, low back pain and radicular pain, unassociated with the characteristic syndrome of that

consult us about other complaints, such as headaches, fits, trouble in the arms, etc., and who only mentioned pain in the lumbar spine or one of the legs when questioned; they had thought that "that" was part and parcel of their particular disability and it had never entered their heads to seek medical advice for that alone.

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4. The neurological disease must not *per se* include the unreliability of the case history (psychic disturbances).
5. Until the paresis and ataxia had become manifest, the patient must have been free from pain.
6. The paresis and ataxia must have existed long enough for pain to develop as a result of the overburdening of the frame. (At least two years?)
7. The neurological clinical picture must not produce analgesia preventing the patient from experiencing pain.
8. The substrate of the neurological disorders must not be seated in or near the root system to which we referred the pain of which the patient complained.
Lastly, is the incidence of these disorders significantly greater than among healthy people?
After drawing up point 8, we realised to our astonishment that a simple, uncomplicated, fresh hernia nuclei pulposi lumbalis affords the finest example of a vicious circle which the pain produced by pathological movements implies! (Through points 2 and 8, nuclear retropulsion cannot, of course, meet the conditions formulated for the demonstration of this phenomenon.) From that moment it was patent to us that the ostensibly simple mechanism of disc herniation is not to be explained merely in terms of root compression. We regarded this discovery as a further challenge to pursue our researches.

It was found that the eight conditions set down reduced our neurological material to two groups, *viz.*,
A. Multiple sclerosis.
B. The after-effects of an operation on the brain or of a cerebral trauma which had led to paresis and ataxia, the patient having survived at least long enough for the secondary disorders under consideration to have developed after sufficient mobility.

Before proceeding further, we should like to dwell for a moment on a very striking example taken from group B, which calls for special comment:

A hairdresser of 52 had always performed his professional duties – which impose considerable strain upon the frame – without discomfort or pain in either back or legs After suffering from headaches for a brief period, he was admitted in a psychotic condition to a psychiatric institution. While he was there, his left leg and arm became affected with a mild form of paresis. Having been transferred to the neurosurgical department of the St. Ursula Clinic, he was found to be suffering from a fronto-parietal convexity meningioma, which was removed The previously existing paresis persisted for some weeks after the operation, but eventually cleared up, leaving only some weakness of the left leg causing him to walk somewhat hesitantly. At check-ups three months later and a year after that the patient stated that *he had no further complaints*, he was working full time again. Very occasionally his left knee gave way, but he was given a clean bill of health and discharged.
Eighteen months later the patient presented himself for treatment for pain in his

ingly to explore these lateral spaces. We, and other surgeons operating in the St. Ursula Clinic who employed this method wholly or in part, found more and more of the abnormalities we were looking for. Thus the *constitution* we had postulated was confirmed empirically by more or less accidental findings.

Could we now, also along other paths of approach, demonstrate the probability of a constitution of the kind pre-operatively? It would be an immense help if the existence of that constitution could be verified by X-ray. In that case minor complaints in neurotics would be seen in a different light. White and Sweet assume that there is an organic basis to every "psychogenic" complaint of pain, arguing from the premise that the disturbed psyche reacts through aggravation of vague feelings of unease upon a site of least resistance somewhere in the body.

The prognosis, too, and more particularly opinions as to appropriate conservative treatment, would be placed on a sounder foundation if a predisposing constitution could be demonstrated from exact radiological data substantiating the predictions made.

The skeleton has already been photographed times out of number, especially in quest of causes for back and limb complaints. If the, as yet unknown, abnormalities we were seeking were demonstrable as clearly *visible* abnormalities, a causal relationship would have been established long ago, either in medical practice or by statistical proof. Hence the *known* congenital malformations (transitional vertebrae, spina bifida and abnormal placing of the intervertebral joints, etc.) could not be the *unique* aspect of this constitution. We therefore scrutinised the radiological field for unobtrusive signs of this constitution, *i.e.*, not for *mal*formations, but for certain formations of the vertebral column which had been observed before, but, just because they occurred so frequently, were disregarded as being physiological. These (mal)formations of a constitutional nature need only be *predisposing*; they need not cause the complaints. We therefore expected that the radiologically demonstrable manifestations of this constitution would differ only *slightly* from the normal aspect of the vertebral column.

The surprising clarity with which some at least of the manifestations of this constitution were exposed by X-ray (see Part III, p. 448) provided us with a fitting keystone for the build-up of our enquiry.

Provisional Conclusions from Findings at Re-operation

The following points may seem to weaken the foregoing reasoning. If this predisposing constitution did in fact exist:

right leg at a demonstration held for neural therapy. When we examined him for this complaint the Lasègue test on the right was slightly positive, the tendon reflexes were the same on both sides (!), the sole reflex on the left was indifferent, sensibility was undisturbed, there was no evident difference between the force of the muscles on each side and the back moved normally.

Just as the neural therapist had been ignorant of the fact that this patient had undergone an operation for meningioma eighteen months previously, so were we unaware, six months previously, that the patient was then already having pain in his right leg.

It is necessary to bear in mind that by no means all patients spontaneously mention "minor" complaints. They think it would "seem fussy" to complain of so unimportant a thing as slight pain in the back alone or in the back and leg. Psychologically, of course, these minor complaints are submerged in the far more vivid impression made upon the patient by the major operation on the brain. Consequently, if the patient has not been specially questioned about them, we do not attach any value to the absence of particulars of these "minor" complaints in old case histories.

Presumably, few practitioners examining patients after operations on the brain will have enquired as to whether there are or have been any complaints of back trouble and radicular pain. If one does so enquire, however, as has now become our practice, one cannot fail to be struck by the frequency of the affirmative answer. *Yet it is not invariably in the affirmative!* Why should the syndrome we are considering develop in one patient presenting slight pathological changes in posture, whereas the same disorders do not produce it in another? Apart from incidental factors, it had to be assumed in a number of cases that a given patient was predisposed by the nature of his endogenous structure to this low back pain and radicular pain. Looking back, we saw that many of these vague syndromes – for which no aetiological cause, such as a hernia nuclei pulposi, was known – had to be referred to compression of the lateral two-thirds of the root and ganglion. We also saw that this compression could be brought about or aggravated by a diversity of pathological movements and changes in posture in the case of *some* individuals. Obviously, therefore, we should look for this predisposing constitution in a previously existing narrowing of the spaces in or around the intervertebral foramen. Moreover, space within the foramen may have been reduced by long antecedent (*e.g.*, congenital) changes or additions to the contents of that space. Our quest was therefore directed to changes like thickening or anomalies of ganglia, roots and dural sac which, if found, would represent the predisposing constitution to which we have referred.

Our *method of operation* was developed along lines which enabled us increas-

later if no spare space has been made and the patient is therefore not allowed to move, or has very restricted movement;
not at all if the space around the root or in the foramen was already limited before the operation and continued to be so after the operation. While the patient's pain is unremitted (recurrent), the root becomes firmly adherent.

To our mind, a permanent root adhesion is all the more apt to occur if space becomes restricted post-operatively, or an antecedent restriction persists. The latter will certainly be the case if a predisposing spatial restriction existed beforehand. There will be a similar state of affairs if a hernia was incompletely removed at the first operation; but in that event a *genuine* recurrent hernia nuclei pulposi is found at re-operation.

The fact that the post-operative patient sooner recovers the ability to move normally (see p. 630) proves that it is often desirable or necessary to provide spare room by widening the entrance to the intervertebral foramen.

The Coincidence of Psychic Disorders with Complaints of Low Back and Radicular Pain

We instituted an enquiry to discover how many of the patients not entirely relieved of their complaints after the removal of a true hernia nuclei pulposi were assumed to be suffering from a psychic disturbance. The figure was found to be 32% (24 in 75 case histories). Only 5 of these 24 patients had been *known* to suffer from mild psychic disturbances before the operation (6.6%).

Although this proportion of 24 to 75 is more or less what we expected, we cannot attach much value to this enquiry. We made it a point to find out how often the various examiners at post-operative polyclinical check-ups had made a note of the fact that they considered after-effects of a psychogenic nature to be present. Such a thing, we thought, could scarcely be established at what is often a single polyclinical examination. These various examiners may have been guided by totally different criteria when forming their opinion. Practitioners who are unable to find any plausible explanation for complaints from a physical examination are all too prone to ascribe them to a psychogenic disturbance. Moreover, if there was evidence of psychic changes, we doubt whether a well-considered differentiation was made between those responsible for and those resulting from the after-effects under discussion.

Psychic *changes*, which are physiological reactions to unpleasant experiences or pain, however caused (restlessness, a tendency to become depressed, irritability, slightly hypochondriacal preoccupation with the affected part of the body), should be distinguished from those particular psychic *disturbances*

1. Why were its manifestations not detected at re-operation?
Re 1. They have been observed by others increasingly, as reports in the literature from time to time show, and by ourselves since we have been on the look-out for them.
2. How could there then have been any question of "negative" exploration in relevant cases?
Re 2. The term "negative exploration" implied that nothing abnormal was found in the intervertebral discs. It was not until after 1948-1949 that we began to search systematically for other abnormalities in the spinal column and, indeed, found them.
3. What transpired at operations for recurrency? Put differently, was it found at re-operation that recurrent complaints were due to manifestations of this particular constitution not noticed before, untreated or untreatable?
Re 3 This question can, we think, be answered in the affirmative.

Note. The causes of recurrency, in so far as they are discovered at re-operation, will be discussed fully later (see p. 624). Suffice it to state at present that the findings lent firm support to the views set out in the preceding pages. Let us cite post-operative root adhesion as a single example. As a preliminary, however, it should be pointed out that only the condition of the field of operation on the same side and level as at the first operation is pertinent to an assessment. The discovery at re-operation of abnormalities at some other level or on the other side merely points to circumstances relating to diagnosis, the technique of operation and, possibly, to a superadded disease.

As we shall show, a recurrent hernia nuclei pulposi at the site of the original one removed on an earlier occasion is exceedingly rare. It has been stated several times that the cause of recurrent radicular pain was the adhesion of a root to adjacent material; this, in our opinion, occurred post-operatively, for no mention was made in the report on the previous operation of anything unusual about the root, though it was quite often stated that, after certain manipulations, "the root is now mobile", or "the root now runs freely". This notwithstanding, the root may be primarily adherent (in a field of operation which has not yet been explored), instances of which will be cited later.

Why should there be root adhesion after an operation in one case and not in another? It is our view that *every* root remains embedded in blood after an operation, is surrounded by scar tissue and "adheres". Through movement, the root will be released post-operatively
soon if the technique employed permits the patient to become mobile at an early date;

It transpired that even the most peripheral rhizotomy in the root sheath was capable of eliminating organically caused radicular pain. This seemed to show that in some way (functionally or organically) the root was involved in the production of pain. As we then saw it, this provided a sufficient reason for assuming that the ultimate seat of the pain which disappeared after the rhizotomy was peripheral to the site of section; for other reasons it appeared to be more definitely circumscribed to the lateral two-thirds of the root and the ganglion at the intervertebral foramen.

Low back pain and radicular pain, without any sign of a disc lesion, were apparently liable to be associated with deformities and malformations of the lower extremities and with neurological disorders accompanied by paresis and ataxia in a mild degree. Indeed, it seemed probable that the complaints were provoked by pathological movements in the intervertebral foramen. Under otherwise like circumstances, only *some* individuals were subject to this syndrome.

A tentative investigation was conducted for the purpose of adducing evidence of the existence of a correlation between the incidence of low back and radicular pain, on the one hand, and certain psychic disorders on the other.

What, then, is the characteristic of these individuals whose foramen narrows so easily, or was already narrowed, and whose movements, as the result of slight ataxia or limitation of motility, are so unco-ordinated as to place an insupportable burden upon the frame? By what could we recognise patients who have become subject to barely controllable and somewhat inexplicable (residual) complaints through the activation of a latent psychic disturbance? If, under otherwise identical conditions, disorders arise in some patients and not in others, it would seem that our enquiry leads us to postulate a definite constitution.

We hope to be able to show in the following Parts that such a constitution does in fact exist and that there are sound reasons for calling it a degenerative constitution.

It must have become clear to the reader – and it may be apt to refer once again to the title of Chapter 2 – that *Part I* of this study is concerned with a groping, as it were, without much support from objective observations or facts reported in the literature, towards views which might possibly suggest an explanation for some, at least, of the complaints which had hitherto seemed inexplicable. That is why we avoided making any mention of exact findings and data recorded in the literature which was not available to us at the time of the development of those views. The purpose of *Parts II* and *III*, on the other hand, is to adduce evidence wholly or partly substantiating them.

which are indicative of a disharmonious development of the individual. Those afflicted in the latter way behave quite differently from persons subject to the former changes, making the impression upon observers as being people handicapped by a labile personality as the outcome of miscarried psychosomatic integration. Their behaviour appears to be psychopathic or neurotic. Not feeling competent, ourselves, to make this finer distinction in our patients, we resolved to make it a practice *to enlist the co-operation of a psychiatrist* for the purpose of recording the incidence of psychic disturbances.

Our provisional conclusions from the tentative investigations *up to that time* can be formulated as follows:

(a) For various reasons it may be that too little attention is paid, before the operation, to the psyche of the patient suffering from low back and radicular pain.
(b) A latent tendency to psychic disturbances may become (temporarily) manifest during the first few months after the operation.
(c) If the organic lesion has not been removed in its entirety, this latent tendency towards psychic disturbances may become active.
(d) There exists a definite correlation between the retention of residual complaints and psychic disturbances.

It is not known at present whether (latent) psychic disturbance favours the retention of residual complaints, or whether organically caused residual complaints bring a latent psychic disturbance permanently into the open.

Summary Part I

While the extirpation of a hernia nuclei pulposi brought complete relief or considerable abatement of pain to many patients, the same intervention failed to achieve the desired result in other cases.

The arguments, based on findings at re-operation, which were advanced to account for this were not always satisfactory. Furthermore, there were some sufferers from low back pain and a radicular syndrome who displayed no signs of an intervertebral disc lesion at clinical examination, nor was such a lesion found at operation. It was unreasonable to ascribe all these syndromes to the result of psychic disturbances.

It became pertinent to enquire whether there were other, unknown, organic disorders which simulate the over-all picture of hernia nuclei pulposi.

Construing from previous experiences at operation, we decided that some negative explorations for hernia nuclei pulposi (as a decompressive, not merely as a suggestive, intervention, be it understood) led to the disappearance or diminution of the complaints, whereas others did not.

PART II

SOME RESULTS OF POINTS OF ENQUIRY SET FORTH IN PART I

THE LITERATURE

Addendum

In the chapters that now follow we shall describe exact observations only if we are able to state the source and the numbers of case histories, operation reports and letters from patients giving details of their subjective post-operative findings. If we are drawing hypothetical conclusions from plausible arguments which cannot, however, be regarded as furnishing proof, we shall say so.

The investigations which form part of the basis of this study were begun as early as 1943. With liberty of movement restricted by the war, many a drear evening was spent in assembling statistical particulars which, even then, were available in large numbers in the St. Ursula Clinic. The writer took up the enquiry at that early date because he had been unable, at the very outset of his training (1942), to share the views then held respecting the mechanism of hernia nuclei pulposi; more particularly, he felt even then that an acceptable explanation was lacking for those cases in which no hernia was found despite the contrary implications of the clinical picture

The aim of the first part of our enquiry was to demonstrate the importance and promising results (as they were considered to be then) of the surgical treatment of hernia nuclei pulposi lumbalis. At that time a "slipped disc" was a fairly new affliction and aroused keen interest far and wide. It is a curious turn of events that those same particulars should now serve to show how little progress had then been made with the problem surrounding the hernia, in fact to point the argument that hernia nuclei pulposi as the cause of back disorders is not as important as it was formerly held to be.

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CHAPTER 3

AETIOLOGY OF THE WEAK BACK

Discussion of the literature on the aetiology of endogenous, morphologically demonstrable weakness of the back and its consequences

By an "endogenous condition" or "tendency" we imply one determined by heredity, as against "exogenous", meaning acquired as the result of extraneous causes; and independently of these terms we recognise "congenital" which, of course, signifies present at birth.

In some cases it is not known whether a disorder is endogenous or exogenous in origin; in others, it arises from a mixed cause, *i.e.*, there is both an endogenous and exogenous factor. An endogenously caused disorder may entail certain abnormal mechanisms which render the body susceptible to the influence of exogenous pathological factors. Seen from this point of view, the aetiology of a disorder is pluridimensional and, therefore, to analyse that aetiology it is necessary to take various factors into account. These can be separated into factors determined by hereditary disposition on the one hand while, on the other, the exogenous causes can be subdivided into necessarily pathogenic factors, predisposing factors and precipitating factors.

These discrete groups of factors will, as they interact, create the conditions in which, or due to which, a disorder arises. Through the interplay of the factors, the conditions will vary from individual to individual.

A trauma so violent as to cause a lesion or fracture, whatever the disposition of the subject may be and in the absence of predisposing or precipitating factors, furnishes a good example of a necessarily pathological trigger. At the same time, it is easy to realise from this example that a less severe trauma may have a necessarily pathological effect upon some particular individual less sound in constitution.

Predisposing causes are to be found both endogenously in the hereditary make-up and exogenously, the former being self-evident. As an example of an exogenously predisposing cause we can take osteoporosis as the result of an internal complaint, owing to which a fracture may ensue from even a minor trauma.

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depressive states and those pathological mechanisms, like pathological motor-activity *, which are psychic in origin.

The foregoing might, we think, be clarified by a mechanical example. Thus, in stress tests it will be found that, under otherwise identical conditions, a linen thread will break sooner than a nylon one of equal thickness. But, however thick this nylon thread may be, as increasing strain is put upon it the moment will come when it must necessarily break. It may, however, occur that a thick nylon thread which has been weakened by sterilisation breaks sooner than a thinner linen thread. Hence, if one is aware of the weakness of some particular material and therefore ties it with special care, the weaker material may conceivably be quite serviceable and be less apt to break than the other which is reputedly stronger and expected to stand up to tougher conditions.

While we intend to deal more especially with the endogenous factors which activate back trouble, it will be necessary also to consider exogenous factors.

Although the literature on endogenously induced disorders is vast, the causal relation between endogenously determined disorders and complaints of back pain is mentioned less frequently than one would have expected. Respecting the most common clinical pictures involving back trouble we have concerned ourselves only with views expressed in a few textbooks and monographs; but we have tried to give a comprehensive survey of the literature on the less familiar clinical pictures. During our search it became apparent that some disorders have almost certainly not yet been reported on in the literature.

We shall deal successively with various parts of the supporting frame of the spinal column, the spinal column itself and the contents of the spinal canal. The placing of the various clinical pictures in the subdivisions of this chapter is not a random arrangement. Our main concern in this study has been clinical functional relations liable to give rise to back trouble, rather than rigid anatomical relations Some disorders involve more than one part of the supporting frame; these will be considered in conjunction with the organ most affected by contingent functional disturbances.

The Muscles

The muscular system contributes roughly 45% to the weight of the human body. The most vital functions of this system are maintenance of body temperature and regulation of carbohydrate metabolism, while its most conspicuous function is that by which the body is enabled to carry out movements.

* This will henceforth be termed "motoricity"; see Chapter 5, p. 221.

No less important is the maintenance of posture by continually changing muscle tension adapted to the needs of the moment. In this respect the muscular system forms part of the supporting frame, especially those muscles which are situated around the vertebral column and pelvis. By equiping the muscles with the necessary motive power, the nervous system plays an essential part in the proper functioning of the frame. With diseased muscles or nervous system, movements may be improperly carried out and abnormal postures may become established.

We set out to ascertain the extent to which affected muscles are responsible for the production of pain, more especially in the back, drawing up a simple plan, as set forth below, in which some of the less common diseases are briefly considered. With this plan as our starting point, we proceeded on a far-flung search of the literature. The results relative to the occurrence of pain in association with muscular disorders are reported within this framework.

Roughly speaking, the functional movement of the muscles can be disturbed by:

1. Atrophy, aplasia, anomaly.
2. Neurogenic and myelogenic disorganisation.
3. Diseases of the muscles.
4. Myalgy, myopathy.

Although we were primarily interested in endogenous affections of the muscles, a subdivision into endogenous and exogenous causes was found to be impracticable.

1. *Atrophy, aplasia, anomaly*

Atrophy of the muscles may result from atrophic inactivity. Let it be added that an inactivity can derive from the patient's hereditary disposition, a striking example of which is the asthenic constitution, which manifests itself as a dystrophic musculature.

Senescent atrophy. This is partly atrophy due to inactivity, though trophic disturbances, such as arteriosclerosis of the vasa nervorum, may play some part in it. These symptoms of old age, which are due to an endogenous factor, do sometimes occur prematurely.

Myelogenic atrophy. Diseased anterior horn cells (or the motor cerebral nuclei) cause spinal nuclear atrophy of the muscle.

Neurogenic atrophy. Disease of the anterior root or motor nerve produces a neurogenic peripheral atrophy of the muscle.

Myogenic atrophy. The anterior horn, anterior root and peripheral nerve remaining intact, the muscle itself is responsible for the atrophy. It is then

necessary to consider whether the cause of the disorganised muscular metabolism resides in the (central) autonomic system.

Atrophies of endogenous origin cannot be distinguished from those caused exogenously by inspection under the microscope, but the myelogenic and neurogenic are microscopically distinguishable from the myogenic. Thin muscle fibres are found in the former, without nuclear multiplication, but with some degree of proliferation of tissue, which may give rise to fixed contractures. The myogenic atrophies, even considered by themselves, are recognizable by the variety in size of the different muscle fibres, their arrangement and dichotomy, possible nuclear multiplication and a certain colouring of the sarcoplasm. (Heidenhain 1918, Adams, Denny-Brown and Pearson 1953).

Congenital muscle defects, dysplasia or aplasia are comparatively rare. Remains of the atrophied muscle are always found in myelogenic and neurogenic atrophy of the muscle, but with aplasia the muscle sheath is entirely void; there is connective tissue, vascular and nerve tissue, but not even a rudimentary trace of the muscle.

Occasionally, one of almost any of the muscles in the human body has been discovered to be lacking. Most commonly it will be the muscles of the shoulder-girdle; far more rarely the muscles of the pelvis and lower extremities. The anomaly is often associated with congenital malformations elsewhere in the frame, but it is well worth knowing that, upon examination, the corresponding segments of the spinal cord were found to be normal. (Bing – Kinnier Wilson, p. 1467, Green, Obersteiner.)

In anencephalic and amyelinate subjects the muscles develop normally and independently, without a central nervous system; in muscle aplasia the nerve cells and nerve fibres appear to grow normally in spite of the aplastic peripheral musculature (Bauer and Bode, p. 262, Kinnier Wilson, p. 1468). Due consideration will have to be given to this fact in an appraisal of the constitutional motoricity of the body and the development of this mechanism on an anthropogenic foundation. Finally, there are muscular malformations which, on account of their shape (hypertrophy, sclerosis, dysplasia), compress or irritate peripheral nerves Yeoman and Vinke compared the topography of the plexus cervico brachialis in the case of the scalene syndrome with that of the nervus ischiadicus in its relation to the superimposed M. piriformis. They surmised, but could not prove (Bradford and Spurling) that this muscle contracted spastically as the result of arthritis of the ilio-sacral joint. They thought that the nerve would consequently be compressed in 10% of the cases. They incised the M. piriformis and reported anomalies found in this muscle. Mention was also made of the fact by Lewin (p 66), with the added comment, however,

that a piriformis syndrome cannot explain a *monoradicular* pain, because the whole peripheral nerve would be compressed by the muscle.

Ober and Smith incised the aponeurosis of the M. tensor fasciae latae. Freiberg accounted for the satisfactory result of this procedure by the fact that, after the incision of the fascia lata, the M. piriformis lying below had greater freedom and exerted less pressure on the sciatic nerve. Neither method found favour for the relief of sciatica.

Bradford and Spurling (p. 118) stated that, *with the exception of some morphological anomalies*, no pathological conditions are known which cause contracture of the fascia lata. Hence the inference is that an endogenously caused contracture of muscle and/or aponeurosis of the fascia lata may exist in some rare cases.

Leigh describes a spastic contracture of the M. obturatorius internus. By a comparable mechanism, pain might be produced in the pelvis and referred to the leg through pressure on the nerve plexus (?) or nerve stems.

2. *Neurogenic and myelogenic disturbances*

Neurogenic disturbances. These causes of muscular disorganisation will usually be exogenous. *E.g.*,
Residual conditions of poliomyelitis anterior acuta;
residual conditions of poliomyelitis anterior chronica (alcohol, syphilis) in which an endogenous factor is also involved;
residual conditions of polyneuritis;
residual conditions after injury to a peripheral nerve.

A myelogenic affection of the muscle may be caused both exogenously and endogenously. Trauma and infectious disease are examples of the former.

Every endogenously induced disease of anterior horn, anterior root or peripheral nerve can obviously likewise be interpreted as an endogenous factor in a causal relation to muscular disability which results therefrom. Such are:
Poliomyelitis anterior chronica, in so far as not referable to syphilis or abuse of alcohol;
syringomyelia;
amyotrophic lateral sclerosis;
the myelodysplasia of Fuchs;
the disease of Tooth-Marie-Charcot-Hoffman and Sotas-Déjérine.

3. *Endogenous diseases of the muscles.* From among these we shall take:

a. Dystrophia musculorum progressiva. The muscles of the back and pelvis

are liable to become so dystrophic that the patient is unable to raise himself; with the bone type (Leyden-Mobius), paresis may occur as the result of atrophy, despite pseudo-hypertrophy of the calf muscles through subcutaneous accumulation of fat. When the paresis reaches an advanced stage, the patients discover new mechanisms by which the still healthy muscles are made to do the work of the atrophied ones, with pathological motoricity as the result.

b. Myotonia congenita. Thomsen's disease.

c. Curschmann and Steinert's dystrophic myotonia.

d. Myasthenia (see p. 44).

The patients under *b* and *c*. have difficulty in carrying out certain movements; they are unable, especially after rest, to straighten out for a long time (post-contraction of the muscles). The normal interplay between agonists and antagonists is disrupted. The patient adopts a shuffling gait.

Endogenous muscular diseases are comparatively rare, both in number and variety presented. This is all the more remarkable in that almost half the human body consists of muscle tissue. This tissue is higly differentiated. Bauer and Bode (p. 262) state with emphasis that the muscles are so vitally important as biochemical organs that individuals suffering from a greatly disorganised muscle system would not be viable.

4. *Myalgia and myopathy*

Fibromyositis (myogelosis). This disease, which is so often diagnosed in medical practice, is seldom mentioned in medical literature. According to Hoelen, it is non-existent and is merely a diagnosis of embarrassment.

Veraguth and co-workers say that myogelosis should not be identified with muscular rheumatism (p. 118). Lewin asserts that the pathological anatomy of myogelosis is not known. He calls it a gel-like change of muscle tissue resulting from changes in the colloidal proportions. Strong (quoted from Lewin, p. 391) refers to a myospastic syndrome. Zukschwerdt (p. 174) writes that primary muscular rheumatism is not distinguishable by a characteristic pathological anatomical picture, but that it does present palpable changes in muscle tone and nodules, particularly proximal to the attachments of the tendons. Armstrong (p 153) states: "The aetiology, pathology and indeed even the existence of so-called 'fibrositis' or 'fibromyositis' is uncertain... No local pathological changes in the affected muscles "*(nodules)*" have ever been observed although the condition is believed to be due to oedema or mild inflammatory changes somewhere in the muscles themselves."

It has also been stated that small soft places (*dimples*) can be felt on palpation, but in both cases the adjacent muscles feel taut.

How are we to account for the fact that some clinicians are able to palpate an abnormality, yet at biopsy find neither nodules nor dimples? Could the answer be that the cause of the palpated abnormality evokes the same pathological findings as at experimental excision? This postulates the possibility of the rupture of a muscle in a small separate bundle, caused perhaps by abnormal muscle tension. This would explain the surrounding painful muscle tension, the flaccid prominence (nodule and/or dimple), the negative pathology and the acute onset. In that event, fibromyositis might well be regarded as a palpable manifestation of that classical complaint "lumbago".

Lumbago is the name given to the acute onset of often chronically recurrent pain in the lumbar region. It is ascribed to muscle rupture as the result of overtaxing or muscle tension. The haemorrhages and the rupture itself are not observed. The condition being highly sensitive to the effect of chill and damp, it is apt to recur, probably in association with renewed muscle tension due to mild neuritis (Veraguth, p. 121).

Myositis is an exogenous cause of muscle weakness and muscular complaints. It will be enough to refer to the full description given of it by Kinnier Wilson (Part I, p. 439), adding only that he describes myofibrositis as a disease, not of muscle fibres, but of the connective tissue which occurs everywhere in the human body, also between the muscle fibres. Apparently fibrositis is microscopically demonstrable: a sterile serofibrinous exudate is absorbed, while there is proliferation of fibroblasts. Nodules of tissue are said to be felt on the tendons, muscle fasciae and aponeuroses, as also deep in the muscle belly. It is emphasized that myositis as such never really occurs by itself, being invariably associated with disorders in other organs (vascular system, skin), as has likewise been described elsewhere (Rimbaud).

Myositis siva: myalgia epidemica acuta (Bornholm's disease) is accompanied by fever and pain in the chest and abdominal muscles; hence it is easily differentiated from the causes of low back pain (Sillevis Smitt, p. 3018, Veraguth, p. 117)

Myalgia might arise in part of a muscle; this phenomenon is mentioned by no-one in the literature except Gordon. In addition to the myopathies already listed we may mention *Endogenous* (congenital and hereditary) *generalised sclerosis of the muscle*, a rare disease forming an endogenous counterpart to myofibrositis (Boeters, Cordier, Löwenthal, Rademecker, Van Bogaert); then *Myodysplasia fibrosa multiplex*, likewise a degenerative condition of the fibres, which is also congenital, but less generalised. The limbs are affected by pseudo-ankylosis of the joints as well as by changes in the muscles. It is also called Arthrogryposis multiplex, Multiple angeborene Gelenkversteifung (Bodart,

Weil and co-workers), Angeborene Gelenkstarre (Gruber, Swalbe, Valentin).

Falling outside this framework there is the discussion of localised pain resulting from Chronic Contractures ("Contractures des artheromateux"; residual conditions after trauma, poliomyelitis anterior acuta, Little's disease, etc.) These pains are brought on locally by stretched tendons, fasciae and, possibly, bundles of muscles; also, in a state of exhaustion, as the result of disorganised nourishment of the muscles. (Sillevis Smitt: crampi musculorum)

In the chapter dealing with the consequences of disturbed muscular function we shall consider whether these contractures may lead to the development of a mechanism which is responsible secondarily for changes and complaints elsewhere in the body

To satisfy ourselves as to whether the above muscular disorders are directly or indirectly responsible for aches and pains, more especially in the lumbar region, we searched for references to the matter in the publications of Armstrong, Biemond, Boeters, Bradford and Spurling, Brouwer, Cossa, Hoelen, Kinnier Wilson, Leriche, Petit-Dutaillis and De Sèze, Schmorl, Sillevis Smitt, Sleeswijk, Thurel, Veil and Sturm, Veraguth, White and Sweet, etc. Nowhere did we find it stated that muscle weakness, atrophy or hypertrophy of the muscles causes pain. Here and there mention was made of excessive fatigue after the slightest exertion.

For all that, complaints of pain are constantly being associated with the disturbed muscular function, *i.e.*, movement and maintenance of posture. These back complaints – weakness, fatigue, pain – are thought to be caused by failure of functional co-ordination of the muscles and its resultants, such as scoliosis and so forth. Schmorl (p. 231) dwells on the relation between atrophy of the muscles and postural anomalies (scoliosis, kyphosis, lordosis), referring to the occurrence of back complaints in association with postural changes. Lubberhuizen and Van der Meer (Sleeswijk, p. 636) detect the same relationship, though the latter lays more stress on the painful spasticity of the soft parts. Van Assen (Sleeswijk, pp. 542 and 586) points out that in advanced age the muscles contain more water. The muscles become less hard and more liable to tear while the quantity of muscle tissue also diminishes, whereas the connective tissue increases The muscles are less powerful. Normally this change in (joint) muscles (and bones) begins to be manifested in adult life, but may be presented early in life if the subject is constitutionally predisposed to this change. The response to decreasing muscular strength and slackening ligaments is a changed posture which may produce pain in the loins and elsewhere in the back. In neurogenic disorders affecting the muscles, the associated pain is ascribed to a pathological condition of the nervous system, such as neuritis.

Summing this up, we may say that myalgia, myositis, traumatic muscle

lesions and, possibly, contractures produce local pain. Apart from these local disorders, mention is seldom made in the literature of pain brought on by diseased muscles, but when it is stated to occur, it is thought to be referable to the disturbed muscular function, *i.e.*, the complaints are ascribed to a changed posture or pathological motoricity.

Endogenous Weakness of the Ligaments

Many ligaments surround the vertebral column, their functions being to hold its component parts together and to provide the necessary flexibility for the muscles to perform their tasks. The ligamentous apparatus is of mesenchymal origin. Pathological changes of the ligaments belong as such to diseases of the connective tissue. Such diseases are often said to be systemic, because the disorders occur simultaneously in several places in the body. Localised affections of ligaments are rare.

It is perhaps because the anatomy of the ligaments is erratic that it is difficult to know if some particular tendency is an anomaly or not. There are well-known examples of this. Thus the course of the transverse tendon of the musculus peronaeus is different in the Negro from that in the European. Larmon (p. 893) carefully dissected ten random vertebral columns and, apart from other variations, found in three cases an abnormal arrangement of ligaments through which the roots and nerves at the site only just missed being compressed. Magnuson (p. 878) reports an anomaly in a ligament which covered the intervertebral foramen exteriorly, as the result of which the nerve was under pressure.

Disorganisation of the plexus of connective tissue, more especially of the ligaments of the vertebral column, may be:

> endogenously congenital,
> endogenously latent;
> endogenously "subclinical" (Kits van Waveren, p. 878);
> endogenous and exogenous through premature senile decay;
> exogenous as the result of disease or injury.

This in itself shows that disturbances of mixed aetiology may arise; it also accounts for the fact that these conditions are variously described, depending on the author's main preoccupation. Research workers in heredity, like Bauer, Bode and Boeters, the radiologist Schinz and the anatomist Schmorl, are primarily on the look-out for the anatomical relations and pathology. The psychiatrist and, in a sense, the specialist in internal diseases are more likely to stress the concomitant psychic disturbances.

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For all that, complaints of pain are constantly being associated with the disturbed muscular function, *i.e.*, movement and maintenance of posture. These back complaints – weakness, fatigue, pain – are thought to be caused by failure of functional co-ordination of the muscles and its resultants, such as scoliosis and so forth. Schmorl (p. 231) dwells on the relation between atrophy of the muscles and postural anomalies (scoliosis, kyphosis, lordosis), referring to the occurrence of back complaints in association with postural changes. Lubberhuizen and Van der Meer (Sleeswijk, p. 636) detect the same relationship, though the latter lays more stress on the painful spasticity of the soft parts. Van Assen (Sleeswijk, pp 542 and 586) points out that in advanced age the muscles contain more water. The muscles become less hard and more liable to tear while the quantity of muscle tissue also diminishes, whereas the connective tissue increases The muscles are less powerful. Normally this change in (joint) muscles (and bones) begins to be manifested in adult life, but may be presented early in life if the subject is constitutionally predisposed to this change The response to decreasing muscular strength and slackening ligaments is a changed posture which may produce pain in the loins and elsewhere in the back. In neurogenic disorders affecting the muscles, the associated pain is ascribed to a pathological condition of the nervous system, such as neuritis.

Summing this up, we may say that myalgia, myositis, traumatic muscle

asthenia: the connective tissue is also endogenously too weak in other parts of the body. The results are ptosis of the internal organs, stomach, liver and kidneys; a tendency to hernias and prolapses; deformities at the extremities, *e.g.*, flat feet, kyphoscoliosis; weakness of the blood vessels, *e.g.*, varices, haemorrhoids. The attachments of the ligaments are slack; moreover, the cross-striped and smooth muscle tissue is underdeveloped; there is a weaker reaction to electrical stimulation. The bones are slender and, partly for this reason, the whole build is slender, long-boned, narrow and slim, with drooping shoulders, chest sunken, abdomen slightly protruding. The subcutaneous fatty tissue is underdeveloped, which further accentuates the absence of a normally developed muscular system. The dental tissue, being of inferior quality, favours caries. The manufacture of the blood is disorganised, hence anaemia readily occurs. The exterior changes in form are reflected in the internal organs, *e.g.*, an abnormally long mesentery, short small intestine, long large intestine, small heart with elongated lung, liver and kidney. Such subjects are said to be hypersensitive to infection, more especially to tuberculosis. Since formation of connective tissue must be involved in any process of healing, this might be accounted for by the fact that, with defective tissue formation, the encapsulation of a process would be impeded or retarded. On the other hand, imperfect functioning of the organs will render the subject more susceptible to infection. This is certainly true as regards tuberculosis of the lung, particularly if the patient is naturally inclined to breathe superficially, thereby failing to provide an adequate supply of oxygen to the lungs. Lack of strong fibrous tissue reactions, however, has its advantages, the patient being less liable to cirrhosis and high blood pressure. Carcinoma is also said to be fairly rare in asthenics. It is a striking fact that scars of healing wounds become broader and remain purple for a longer period than in other patients.

The *character* of asthenic patients is apparently endogenously different. They are weak-willed, are neurasthenically inclined and the preponderant mood is a depressive one.

It is pointed out that the intellect need by no means necessarily be of poor quality, but there appears to be a certain incompatability between the inherent potentialities of the mental equipment of these patients and the ability to bring those potentialities to fruition. Given a well-defined task, they are able to get through a good amount of work with grim tenacity, but, once beyond that circumscribed area, they are again assailed by a pathological lassitude. Handicapped with such a character, the asthenic is soon liable to find himself involved in conflict. He is a type that should not be judged only by the aspect he presents under very special circumstances. He may make the impression of

Two detailed reports appeared, one shortly after the other, in the Dutch literature (Kits van Waveren and Rümke, 1955). The curious part about these is that here the physician concerned with internal diseases approaches the problem from the psychiatric point of view, whereas the psychiatrist is at pains to point out that, when confronted with unmistakable psychiatric clinical pictures, such as neurosis in particular, the examiner should without fail pay due attention to the simultaneously present internal disorder. The psychological background of the approach to a problem in the light of a specialisation other than one's own is easy enough to understand; the manifestations within the specialist's own domain are regarded as self-evident and, by virtue of that very fact, obscurities within some other specialised field may be illuminated.

The most common and unambiguous disease of the connective tissue is *Asthenia* (fibrogenesis imperfecta generalisata). Virchow referred to it as far back as 1872. Bennicke called asthenia an anomaly of constitution in 1881. Tuffier (1894) was the first to describe it fully with its concomitant complicating conditions, also mentioning the accompanying psychic disturbances. Stiller described this clinical picture in even greater detail in 1907, again with emphasis on the constitutional aspect. Next, Bruckhardt, Kretschmer and Schlegel fitted asthenia as a constitutional disease into their plan of types to assist in the appraisement of character, temperament and psychoses.

Asthenia is considered to be *imperfect development of the connective tissue within the whole organism, i.e.*, the connective tissue, the ligaments of the skeletal frame, the interstitial connective tissue, the connective tissue in vascular, nerve and muscle sheaths, fasciae and ligaments. The asthenic, fragile spine is recognizable on radiographs by the tall cylindrical shape of the vertebral bodies (especially in the lateral projection). We know by personal experience that aspects of this kind occur locally, mostly in the low thoracic or high lumbar region. This more local expression of general asthenia is accompanied by a wasp waist, hyperlordosis or slight thoracic kyphosis. From the prone position the patients rise by drawing up the upper part of the trunk at an acute angle; their movements are reminiscent of those typical of sufferers from dystrophia musculorum progressiva. Functional listheses are seen on functional radiographs. These patients commonly complain of low back pain.

The incomplete formation of the fibres of the connective tissue is *demonstrable histologically* (Boeters, Hueck, Erbbiologie III, p. 285). Bauer and Bode (Erbbiologie III, p. 284), Friedman, Kits van Waveren (p. 867) assume that there is a central cause for this defect. Its central character is held to be responsible for the simultaneous occurrence of various disorders in those suffering from

overlooked, that in patent cases of psychic disturbance the patients may well be suffering from a somatic disorder. This view is entirely concordant with Bauer's, though reached from a different angle. Asthenic patients are so liable to disturbances in the vascular system, the lungs and so forth that it ceases to be possible to attribute the development of somatic disorders in sufferers from asthenia to mere chance. The cause is to be sought in impaired functioning of the organs: superficial respiration increases the risk of contracting tuberculosis of the lung; from youth up, a hypoplastic heart suffices for a body accustomed to lethargic movement, but soon gives out if suddenly overtaxed.

Pain in the joints and muscles and abnormal fatigue, liable to be felt more especially in the lower extremities and the back, have already been mentioned as *subjective symptoms*. But this syndrome of effort need by no means derive from the somatic condition in the back and legs; it may be the result of an acquired lassitude. This acts as a protective mechanism against all demands which might eventually require the overburdening of substandard organs.

In extreme cases asthenia has been shown to be probably *endogenous* in character; the existence of an endogenous factor is generally assumed in many gradations. Schmorl (p. 230) sees asthenia as the result of endocrine changes of an endogenous nature. He too reports the simultaneous occurrence of other disorders, such as endocrinopathic kyphoscoliosis, myopia and, at the same time, other diseases, such as syringomyelia. In a very large series of dissections he found degeneration of the ligaments of the vertebral column in 15% of the cases, though he does not state whether he regards this as a manifestation of asthenia.

Weakness of the ligaments, of a mixed endogenous and exogenous character, likewise occurs as *premature senility*. An endogenous disturbance is said to be responsible for premature senility as also for a premature climacteric and associated symptoms (De Snoo, Sleeswijk, pp. 602, 604, 607).

According to Lewin (p. 633), sclerosis begins in the ligaments at the age of 20. The ligaments and muscles (as well as other organs) become steadily weaker as the years advance; unless the individual's mentality adapts itself to this state of things, unphysiological conditions arise through strain and, consequently, complaints.

Inactivity may weaken the ligaments, or they may even perhaps be directly weakened by *pathological conditions*, such as infectious diseases and toxaemia. The prognosis is entirely different from that of endogenic disturbances. Recovery soon follows upon appropriate exercising in the same way as in atrophy of the muscles through inactivity; and this is consistent with the experience that the firmness of the supporting apparatus, especially of the

being a vitally alive personality reacting violently in a transient state of excitement, but as soon as the abnormal stimulation subsides, the neurotic asthenic sinks back into his constitutional apathy.

Although there is no sure evidence that asthenia is *hereditary*, it is considered very probably to be so (von Verschuer). The difficulty is that, as extreme forms of asthenia are rare, the opportunities for following its incidence in certain families are attenuated.

The cases of asthenia in which the somatic stigmata fall into the background and the psychic symptomatology predominates are far more numerous. Cohen, Wheeler and Wight estimate the incidence of the mitigated form of asthenia at approximately 5% of the total population. Friedman points out that asthenia may occur "subclinically" and believes that this is the case in 30% to 40% of young people.

Neurocirculatory asthenia, one of the particular aspects of asthenia, has, since it was so called by Oppenheimer and Levinde, acquired the status of a disorder in its own right. Its pathogenesis is regarded as being partly exogenous and partly endogenous. It is said that exogenous factors, especially those taking the form of psychic conflicts, grafted upon an hereditary constitution, start off neurocirculatory asthenia (Kits van Waveren, p. 877), when subjective symptoms occur, like heart complaints of a functional nature, dyspnoea, headaches, dizziness, gastro-intestinal upsets, lassitude and pain in muscles and joints. There are also bodily signs, such as dilated pupils, tremors, diaphoresis, abnormal blenching upon the least provocation, tachycardia, slightly increased body temperature, arthroidal loss.

In this form of asthenia, phobias stand out prominently from the psychic aspects just mentioned. Friedman sees in such phobias an expression of fear of life The abnormal fatigue and intolerance of exertion are in part an expression of the phobia, which has made exertion itself its object.

It should be understood that a distinction must be made between these psychic aspects of asthenia and the neurotic and hysterical forms of reaction to be found in normal individuals

When we assume that neurosis and other mental disturbances, like psychopathy, can only arise from an endogenous basis, this does not exclude the possible occurrence of neurotic reactions caused exogenously in persons who are unable temporarily to withstand the violence of certain impulses Here again there will undoubtedly exist gradations from the normal psyche to the endogenously vulnerable psyche, the latter being more readily impelled to react to less violent impulses. Such mixed forms are likely to present themselves precisely in neurocirculatory asthenia.

Rumke emphasizes the possibility, which, he says, should on no account be

the foramen, he was able to show that the ventro-dorsal size of the intervertebral canal thereby became 2 mm narrower. The diameter of the ventro-dorsal aspect of the *foramen* is something more than 7 mm, while the average diameter of the *ganglion* is found to be 7 mm. Obviously, therefore, any slight change in the ligaments around the foramen will cause constriction of the root. These changes may be brought about by weakening of the ligaments themselves, when they thicken and become relatively too long. If their function decreases, the vertebral bodies may not be held in their relative positions adequately, with spatial narrowing as the result. Finally, secondary oedema of the ligaments might well tend to reduce space. But it is derangement of the *function* of the ligaments, in so far as they contribute to the support of the vertebral column, which is regarded as the chief factor precipitating complaints in the above condition.

Back Complaints in Association with Malformations of the Vertebral Body

Deformities of the vertebral body are common. Malformations due to endogenous as well as exogenous causes are described fully in the literature (in textbooks on heredity, pathology and radiology). In them, however, the clinical aspect, notably the occurrence of associated complaints, is naturally not specially dwelt on; often it is merely stated that the abnormalities are liable to give rise to complaints. The search has to be extended to case reports and monographs, the latter being rare, in contrast to, or precisely because of, the many varieties of malformation. Taking the function of the organ as the focal point, we have divided the malformations into several groups.

The consensus of opinion in the literature is that the disturbances we are about to deal with are endogenous in character. Bauer and Bode (Erbbiologie III, p. 195), Boeters, Schmorl and Schinz review the matter *in extenso*, pointing out in particular that these disturbances should be regarded as different aspects of one and the same entity. It is not that one particular malformation is hereditary, but that "the unimpeded development of the vertebral column is hereditarily thwarted". Some ancestor will have had a spina bifida; descendants will have a cervical rib and/or a spina bifida, and so forth. If something interferes with the original disposition of the vertebral column, the growth to normally formed vertebrae individually will likewise be impeded. The authors assume that a disturbance of the gene responsible for the construction of the vertebral column will involve disturbance of those genes which determine the genotypical aspect of the individual vertebrae. In that event, fissure formation,

ligamentous system, benefits generally from prescribed exercises followed by controlled training. In this context Bauer and Bode (Erbbiologie III, p. 263) again emphasize the importance of the constitution. The asthenic's posture and whole constitution can be improved by physical exercise, but the degree of success depends entirely upon his constitution. A man is born an asthenic or, say, with the potentialities of an athlete. However well thought out the physical exercises and training may be, the endogenous asthenic cannot get beyond the limits of his disposition.

The manifestations of asthenia are so various that they could be discussed under the headings: psychic abnormalities, disorders of the ligaments (as we have done) or equally well, lesions of the muscles. Here follows a note on an affection of the muscles which has to be differentiated from asthenia:

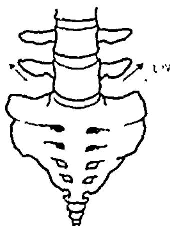
Bartstra (1958, p. 1442) defines *myasthenia levis non-paralytica* as the cause of low back pain (exhaustion of the muscles upon faradic stimulation, hypersensitivity to curare, good reaction to small doses of physostigmine). Several aspects of the syndrome, which only occurs in middle-aged women, resemble the combination of an involutional syndrome and asthenia.

In addition to these more common clinical pictures there exist less well-known endogenic pathological conditions of the connective tissue; and we may be sure that there are also subclinical forms which, maybe, could not even be classified as a definite malady. As an illustration, more than anything else, we might mention *fibrodysplasia elastica generalisata* (syndrome of Ehlers-Danlos), Boeters (Erbbiologie III, p. 227) and Schinz (p. 1360). This is another systemic disease of the mesenchyme. The joints and the skin are too stretchable. The elastic components of the connective tissue are insufficient. This clinical picture presents deformities of the extremities, such as flat feet in a pronounced degree and habitual luxations. There is a strong tendency to herniation and haemorrhagic diathesis is said to be by no means rare. The skin is conspicuously flaccid and appears to be far too loose, while in some places it is very thin. On palpation it feels like rubber (cutis laxa).

The question as to whether asthenia can be painful will be considered under the heading of pathological motoricity. On the one hand the literature refers to pain in the joints and ligaments, on the other, it is repeatedly pointed out that, as the result of this *slackness of the ligaments*, movement, and more especially posture, is affected (Chapchal, Sleeswijk p. 554, Van Assen). These authors describe the associated mechanism as insufficientia vertebrae, or insufficiency of the supporting apparatus.

Magnuson (p. 878) demonstrated by experiment that diseased ligaments are capable of causing radicular pain By injecting saline into the ligaments around

which means that there is a displacement in the cranial direction; in that event the slightly abnormal vertebra directly above the sacrum is also somewhat displaced cranially and probably is, therefore, a lumbarised sacral vertebra. The transverse process of the twelfth thoracic vertebra is attached to the vertebral body at a higher level than that of L.1. This, too, might be taken as a sign showing whether a certain vertebra is thoracic or lumbar. Neither of the latter aids is reliable, however, because all that can be established is that either the 12th thoracic or first lumbar vertebra is slightly abnormal, without knowing definitely whether it is the result of thoracication or lumbarisation, *i.e.*, resulting from cranial or caudal displacement of the vertebral column.



Further, L.IV is recognizable by its slender, slightly upwards tending tranverse process.

Values of roentgenographic width of the L.V transverse process above 19 mm may be taken as evidence of an attempt at sacralisation – even though there is no tendency towards wing-shaped configuration nor any articulation or contiguity with the sacrum (Southworth and Bersack, p. 634).

When Kühne expressed the view that it is not a given anomaly which is heritable, but the tendency of the spinal column to develop anomalies, it was precisely with the transitional vertebra in mind. To underline this he introduced the term "*displacement tendency*". Paired heredity factors control this displacement tendency either cranially or caudally. From vast material Kühne adduced evidence to show that a dominant gene directed the displacement cranially and a recessive gene did so in the caudal direction. An identical disposition may *cause cranial displacement, with cervical ribs and lumbarisation of S.I, in one* member of a family and caudal displacement, with sacralisation of L.V, in another member of the same family.

Rosenberg regards the transitional vertebra as a variant occurring in human ontogeny, stating that a physiological displacement of the vertebral column relative to the pelvis, etc. takes place during embryonic development. Different variants arise because this displacement varies in degree from individual to individual. If, then, the tendency to the formation of anomalies and the cranial or caudal displacement already exists endogenously, the degree of the anomalies and the incidence of many other concomitant phenomena might be controlled by (other) endogenous factors. These in turn are affected by external factors

wedged vertebrae, displacements, distortions and departures from bilateral symmetry are to be considered as accompanying disturbances.

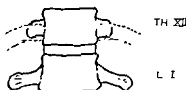
This comes out most clearly in transitional vertebrae which, as a matter of fact, is also an important phenomenon from the functional point of view.

The transitional vertebra

The division of the vertebral column into 7 cervical, 12 thoracic, 5 lumbar and a varying number of sacral and coccygeal vertebrae is considered to be normal. The transition from lumbar to sacral vertebrae is as a rule clearly visible in radiographs, as the lumbar vertebrae lie as it were undetached above the sacrum and pelvis, whereas the sacral vertebrae are fused into a single unit and are attached to the pelvis. This sharp division appears to be arbitrary, however, upon close inspection of the lowest lumbar vertebra above the sacrum for aspects which are intrinsically those of the sacral type of vertebra. This 24th vertebra from the apex is sometimes so similar to a sacral vertebra that the condition is described as *sacralisation*. Conversely, the term *lumbarisation* is used to describe a 25th (first sacral) vertebra which projects far beyond the pelvis and simulates the aspects of a lumbar vertebra. The mere aspects of these 24th and 25th vertebrae cannot reveal whether it is sacralisation or lumbarisation; they should then be called "transitional vertebrae". The true facts can only be ascertained by photographing the whole vertebral column and counting the vertebrae in order to decide whether the 24th is sacralised or the 25th lumbarised. The following are aids, but not entirely reliable ones: Noting the number of sacral vertebrae of which the sacrum is composed, sacralisation is more likely if the sacrum is elongated.



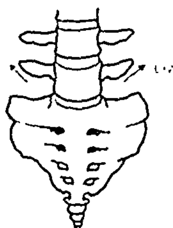




Displacement of the spine in relation to the pelvis in a caudal direction

There may be lumbar ribs at the highest mobile vertebra above the sacrum,

which means that there is a displacement in the cranial direction; in that event the slightly abnormal vertebra directly above the sacrum is also somewhat displaced cranially and probably is, therefore, a lumbarised sacral vertebra. The transverse process of the twelfth thoracic vertebra is attached to the vertebral body at a higher level than that of L.1. This, too, might be taken as a sign showing whether a certain vertebra is thoracic or lumbar. Neither of the latter aids is reliable, however, because all that can be established is that either the 12th thoracic or first lumbar vertebra is slightly abnormal, without knowing definitely whether it is the result of thoracication or lumbarisation, *i.e.*, resulting from cranial or caudal displacement of the vertebral column.



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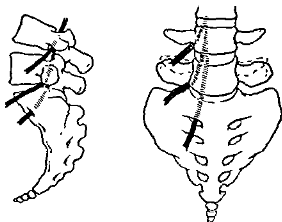
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(Kuhne). Thus variations and anomalies of the nervous system, the connective tissue and the vascular system, which occur in conjunction with the malformations referred to, are likewise of mixed endogenous and exogenous origin.

We learn from investigations made by Abel, Fischer and Kühne that the transition between lumbar and sacral vertebrae lies grossly anatomically between the 24th and 25th vertebrae in two-thirds of the human race. If, however, *attention is paid to anatomical minutiae, such as indications of* aspects of a transitional vertebra in parts of the spinal column or segments of the vertebra, then it appears that:

normal conditions prevail in only	7%
displacements in cranial direction occur in	59%
displacements in caudal direction occur in	34%
	100%

The transitional vertebra in its entirety and in so far as it forms part of the functioning spinal column may best be defined as a vertebra which is less solidly attached than a sacral vertebra and less freely mobile than a lumbar vertebra. This relative mobility and fixation is often associated with abnormally articulated small vertebral joints and with a wing-shaped extension of the transverse process; approximately at the site of the ilio-sacral joints, this forms, either on one side or on both, a ligamentous union, a pseudarthrosis or a true joint with the pelvis (Van Assen, Sleeswijk, p. 554).



The lateral process may be so large that the fourth and/or fifth lumbar root run(s) over it; inflammation of a (pseudo) joint or bursa at the site of contact between the process and the pelvis may lead to or exacerbate compression or irritation of these nerves (Armstrong, p. 114.)

Apart from the occurrence of unilateral enlargement of the transverse process, the literature mentions the asymmetry of parts of the transitional vertebra (Brocher, p. 42). It is also stated that only parts of the vertebra, the arch, the spinous process and the spinal canal may bear the distinguishing marks of a transitional vertebra. Schmorl (p. 59) quotes

the literature liberally, mentioning in particular the triangular sacral shape which the spinal canal already assumes at the site of the transitional vertebra.



Hemisacralisation,
Asymmetrical spinal canal,
Intervertebral *canal* for L.5 root

He also complains (p. 59) of the lack of reliable statistics relating to the transitional vertebra (varying from 0.6% to 26% in the case of 23 investigators).

The intervertebral disc below the transitional vertebra is often no more than rudimentary. It may be of value to take this into account in the differential diagnosis of disc narrowing as the result of nuclear protrusion. (Brocher, p. 40.)



There is widespread agreement on the coincidence of vertebral anomalies and transitional vertebrae with back complaints, *viz.*, Armstrong, Chapchal, Kinnier Wilson, Krayenbuhl, Martius, Ruding, San-Giorgi, Schmorl and many others. Brocher, Cossa, Schmorl state that sacralisation occurs in 25% to 60% of the patients suffering from sacral pain, lumbar pain and back pain. They emphasize that it was not necessarily the transitional vertebra that caused these complaints. They regard the transitional

vertebra as a constitutional factor in association with which an accessory disorder brings on complaints. Such disorders are arthrosis, bursitis, periostitis, static complaints through scoliosis, hernia nuclei pulposi and so forth. Southworth and Bersack (p. 634), however, found no causal relation between transitional vertebrae and complaints referable to the low back in their material.

Schaap is of opinion that there is no direct relation between these pains and the structural anomalies of the lumbo-sacral part of the vertebral column. (He does, however, mention the fact that many sufferers from low back pain exhibit transitional vertebrae.) His opinion is founded mainly upon the absence of neurological abnormalities. He believes that in many cases chronic back pain is due to a static strain, especially if the disposition of the spinal column is deficient. Brocher (p. 42) assumes an endogenous predispositon to low back pain and various radicular symptoms owing to degeneration of the disc above an asymmetrical transitional vertebra brought about by static strain.

We suspected, from some of our own observations to be reverted to (p. 290), that, where there are transitional vertebrae, the *innervation patterns* of the lumbo-sacral roots are aberrant. In the literature this relation has escaped the notice of the investigators; reflex anomalies and variations in the sensory innervation patterns of the roots are generally reported without comment. Schaap supposes that disturbances in development supervened at such an early stage that the expanding nerve tracts were able to adapt themselves completely to the altered anatomical condition. He does not, however, suggest how they contrive to do this. When discussing innervation patterns, especially of roots L.4, L.5 and S.1, Armstrong (p 95) expresses the opinion that anomalies of the lumbar plexus are relatively common, as are variations in the number of mobile lumbar vertebrae. He does not clearly indicate whether there is a correlation between them. Apparently no reliable statistics exist on the incidence of plexus anomalies. He deals in detail with the relationship of the fourth and fifth lumbar roots to the transverse process of the normal and of the transitional vertebra, but illustrates his remarks by a normal root system (p. 116). To this we shall likewise revert. Nuclear retropulsion of the disc between L.III and L IV is said by Kravenbuhl (p 39) to occur more frequently in the presence of transitional vertebrae Again, the fact is stated but not explained, nor is it brought into relation with the function of the spinal column.

Our scrutiny of the literature on transitional vertebrae – so crucial to the function of the vertebral column – may be briefly *summarised* as follows

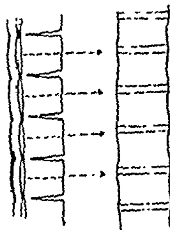
It is generally agreed that transitional vertebrae are very often associated with complaints of pain in the back or legs The authors incline to the view

that it is not the anomaly itself which is responsible for the complaints, but an incidental disorder, predominantly excessive strain through static factors.

Broadly speaking, the same may be said of many other vertebral anomalies. Here we would specially mention:

Fused vertebrae (synostoses), wedged vertebrae, cleft vertebrae

These malformations are also thought to be associated with disorganisation of the original structure of the vertebral column, this also involving disturbance of the genes controlling the phenotypic aspect of the individual vertebrae. Given a deficient spinal column as a whole, local malformations then arise, as the result of which many secondary changes are liable to be precipitated. (Bauer and Bode, p. 192, Brocher, Moppert, Roger and Astier.)



"At a very early stage the spinal cord is to be seen on the back of the embryo, with the rudimentary vertebrae to either side. These primitive vertebrae give off the sclerotomes, from which the vertebral column and the rest of the skeleton are built up. At a given moment the centre of the sclerotome divides into a cranial and caudal part. The *primary vertebra* develops from coalescence of the caudal and cranial parts of two adjacent sclerotomes. The mass of tissue between the cranial and caudal parts becomes an intervertebral disc. The *permanent vertebra* is formed as the result of further coalescence of the left and right primary vertebra and the vertebral arches; hence it consists of four different parts, *i.e.*, the left and right halves, each of these, again, consisting of a cranial and caudal part. With only partial or no division at all into a cranial and caudal segment there arises what is called a *fused vertebra*, which is roughly twice as long as a normal vertebra.

In some cases these fused vertebrae do not grow to their usual height and this may make diagnosis difficult. Failure of or deficient coalescence in the median line produces *cleft*, *butterfly* or *wedged* vertebrae. There are two types of the latter, the large and small wedge-shaped vertebra. In the large type, one half of the vertebra is normal, while the other is only rudimentary. The small type is merely half a vertebra. Sometimes in this case a second wedged vertebra is found in a higher or lower segment of the spinal column, but on the opposite side". (After Schaap.)

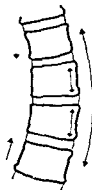
The symptomatology varies with the vertebral level.

According to Klippel-Feil, cervical synostosis is the most familiar anomaly. This disturbance is thought to be central in character because other disorders occur at the same level, such as meningioma and neurofibroma. But the more obvious assumption in that case would be a segmental disturbance.

Combinations with other malformations occur over the entire vertebral column, *e.g.*, also in the central nervous system at levels that may be distal to the synostosis (spina bifida, cervical ribs, scoliosis, kyphoscoliosis, smaller number of vertebrae, exostosis, funnel and pigeon breast, myelodysplasia, encephalocele, hydrocephalus, club-feet, etc.).

It is rare for the deformities to be associated with primary neurological disorders. The patients do not as a rule complain of pain. With increasing age "rheumatoid" complaints occur which, according to the latest views, are due to secondary disorders (arthrosis deformans, spondylosis deformans, symptoms of fatigue as the result of static strain).

The vertebral column may also become distorted by endogenously caused disturbances to growth occurring during the patient's lifetime. We recall two diseases, as an example, which are in a sense complementary. Chondrodystrophy is a result of premature arrest of enchondral osteogenesis. The vertebrae are broad and flat, whereas the intervertebral discs are relatively too thick. This is the predisposing factor which renders the subject liable to disc ruptures (Schreiber and Rosenthal, p. 648). By contrast, the skeleton of those suffering from macrodactyly grows in length too rapidly; the tendinous tissue being unable to keep up with this growth, contractures and malformation of the joints occur. The disease is associated with cardiac and ocular disorders; the psyche is normal but laggard; the patient is highly sensitive to infection and is predisposed to rickets. Low back pain is not specially mentioned (Bauer and Bode, Erbbiologie III, p. 160, Weve, p 1)

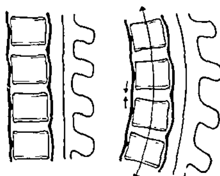


The foregoing endogenously caused malformations are sometimes associated with secondary scoliosis. Scoliosis and kyphoscoliosis, however, can be inherited, without any other accompanying deformities; and this also probably applies to abnormally pronounced or flattened lumbar lordosis, which is mentioned by Faber, Bradford and Spurling (p. 103), Schmorl (p. 232) and Sillevis Smitt. Scoliosis is said to be primarily congenital and hereditary in 5.9% to 30% of cases. These widely spaced figures are eloquent of the difficulty of recognising the cause of scoliosis; nor is it clear whether narrowing, asymmetry and the like were the cause or effect of

scoliosis. Codornu (p. 94) reports congenital essential scoliosis in twins. Heuer is of the opinion that primary idiopathic scoliosis originates in inhibited lateral growth of the vertebral column on one side and unimpeded growth on the other, owing to which the column slants.

It is not yet properly understood whether *adolescent kyphosis* (Scheuermann's disease) is the result of endocrine disturbances, insufficientia vertebrae, osteoporosis or some other basic disease. Rickets can be ruled out as a cause, because the phosphate level in the blood is unchanged (Müller, Schmorl, Wolf, p. 223). The vertebral bodies are anteriorly narrowed and wedge-shaped. "Schmorl's nodules" are seen so often that this anomaly is regarded as a secondary result of changes in the intervertebral discs (Schmorl). The vertebral bodies are said to undergo changes as the result of ingrowing connective tissue, right down to the spongiosa.

Junghanns (Rheumatism Congress 1955) spoke of "disturbances of the growth layers at the basal and alar laminae of the vertebral bodies, owing to which discal tissue can penetrate into these bodies". Through their defectiveness, fine cracks are formed in the cartilaginous plates of the disc; strain causes atrophy and necrosis. The annulus fibrosus is said to remain intact. The disc is liable to disappear altogether, when synostoses may be formed. In early youth this may involve spondylosis deformans. The majority of authors give strain of defective intervertebral discs as the cause of this malady (Boerema, Scheuermann, Schinz, p. 1438, Van Assen). It occurs predominantly in the middle and lowest thoracic and uppermost lumbar vertebrae (Brocher, p. 20). This author stresses the endogenous disposition to low back pain; he says the lumbar form brings on back pain in early life (13th to 30th year), after which the symptoms of cervical arthrosis develop in the same individual at a later age (40th to 50th year)



The *kyphosis of old age*, no less, is determined endogenously. Here, too, the anomalies begin in the anterior part of the disc, but the adult vertebral bodies remain unchanged; *i.e.*, secondary changes might take place which, through ossification of connective tissue proliferation, convert the face of the higher thoracic vertebrae to a single bony entity The height of the vertebral bodies remains constant The forward flexion is brought about by the destruction of the anterior part of the discs and the

wasting and ossification of ligaments and connective tissue. It should here be borne in mind that the anterior longitudinal ligament (as opposed to the posterior longitudinal ligament) runs from body to body (Exner p. 15). A forward flexion results from sclerosis and contraction of these ligaments. It cannot be said that either adolescent or old-age kyphosis is caused by a degenerative disc. It would be more cautious to say that both are associated with a defectiveness – probably due to an endogenous cause – of parts of the intervertebral disc. The invasion of the spongiosa of the vertebral bodies by connective tissue is secondary. This should be differentiated from primary sclerosis, which is the source of *osteosclerosis fragilis generalisata* (Albers-Schonberg's disease). We shall not here deal separately with this so-called "Marmorknochenkrankheit" (Boeters, p. 118, Schinz, p. 664), nor with *polyostitis fibrosa dysplasia* (Albright's disease, Jaffé-Lichtenstein's disease). These syndromes, rare though they be, have been discussed many times in the literature (Prick and Woltering, Schinz, p. 1150, Schmorl, p. 90). Thanks to their specific anomalies on radiographs, they are not difficult to diagnose and they are not likely to be overlooked. They set up local pain; moreover, by impeding the normal functioning of the vertebral column as a supporting frame, they also produce pain elsewhere in the back.

It is apt to point out here that the postural changes discussed in the preceding paragraphs, such as scoliosis, kyphosis and the like, may also result from diseases of the bone tissue. More especially, it is necessary to differentiate between senile kyphosis and the kyphosis which is the effect of osteoporosis, which is more often caused by a pathological process in advanced age than in youth. We are now on the borders of impaired calcium metabolism, the consequences of which so often give rise to complaints, in the low back and elsewhere, that we have to reckon with its possible existence in differential diagnosis Confining the boundaries as much as we can, we should discuss in a general way osteoporosis, osteomalacia and those indications which point to demineralisation of the skeleton. (See page 62)

Postural changes may also be manifested by changes in physiological *lumbar lordosis*. One of the most pronounced is the abnormal lordosis towards the end of pregnancy, to which some of the pain and discomfort of which women complain is ascribed. It provides a striking example, moreover, of a case in which physiological conditions may remotely bring about a changed posture which causes pain. There are a vast number of disorders which may flatten lumbar lordosis The only indication it gives to the practitioner who finds it is that something is wrong with the spinal column. If the complaints were due to an unadulterated psychogenic cause, the normal lumbar lordosis would *not* be

wanting. This does not provide a reliable differential diagnosis. The patient is aware of pain and may react to it with muscle tension; moreover, the complaints may be predominantly psychogenic and meanwhile superimposed upon minor organic disorders. Nor does the absence of a flattened lordosis furnish certain evidence on the nature of the complaints described; in typical organic lesions, such as a hernia (later verified operatively), the physiological lumbar lordosis may be maintained.

The foramina seem to widen upon extension of the lumbar spine. It is difficult to tell whether this posture is assumed to alleviate root compression in the foramina.

Schmorl (p. 231) does not describe an endogenous or congenital change in lumbar lordosis present from the outset.

The exogenous causes may be briefly listed as follows:

Accentuated lumbar lordosis:
- Pregnancy
- Adipose paunch
- Compensation for kyphosis elsewhere in the spinal column
- Chondrodystrophy
- Progressive muscular atrophy
- *Spondylolisthesis*

Neutralised (reduced) lumbar lordosis (Brocher p. 4):
- Disc lesion including hernia nuclei pulposi
- Fused vertebrae
- Trauma and infection
- After laminectomy.



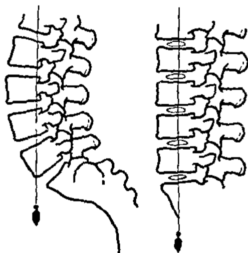
As a result of a very marked lordosis, the spinous processes of two vertebrae, one above the other, may come into contact. This may start off pseudarthritic changes and produce pain in the back; it is Baastrup's syndrome and is sometimes called "kissing spines" (Chapchal, p. 134). Brocher (p. 30) had never encountered this syndrome in any person under the age of 25. In the literature, this anomaly is considered to be highly significant and the physician confronted with rather ill-defined complaints about back trouble would do well to keep it in mind. Its existence should be suspected when hyperextension is attended with localised pain in the back. The anomaly will have to be differentiated radiologically from abnormalities of the vertebral arch, which produce the same local pain upon hyperextension; *e.g.*, a loose arch.

Finally, postural changes, especially *scoliosis*, may be brought about by

various other causes, such as tumours, inflammation, traumata and disc lesions, painful or otherwise.

The postural change due to asymmetrical, painless narrowing of the disc might be considered as purely mechanical; but as soon as the basic cause is painful, contractures and muscular tension are sure to occur, making the sufferer assume the least painful posture. At one moment the scoliosis will be homolateral, at another it will tend contralaterally, this direction being liable to vary with the stage of the malady.

We cannot tell by the direction of the scoliosis in which half of the body the cause is to be found. Painless scoliosis, especially, is apt to change from instant to instant and the sudden deviation to the right or left from the median line is clearly visible (Lewin, p. 504). *Flattened lumbar lordosis* as the result of an organic disease is also a protective mechanism. The median line of the body comes in front of the nucleus pulposus with flexion, and behind it with hyperextension. Through avoiding painful movements, the spinal column will become fixed in the most stable position. (Median line between discal ball-and-socket joint and small vertebral joints.) This position is obtained when the planes cover each other almost completely and that is when, in the presence of flattened lordosis, the vertebrae seem to be stacked one upon the other.

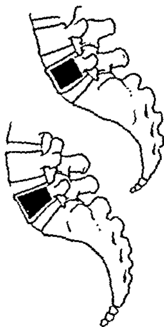


We shall revert to this presently. Scoliosis is often reported to be present in association with Von Recklinghausen's *neurofibromatosis*. We have to assume that this scoliosis is caused endogenously, but it is as yet unknown by what pathophysiological mechanism it becomes established. It might conceivably be the result of a local affection of a vertebra, forcing the spinal column into an abnormal position, or it might be a secondary result of painful muscle strain (Schmorl, p. 230, Prick, Van Wulfften Palthe).

The abnormal attitudes assumed after amputations from the lower extremities and paralysis of the nervous system afford an excellent example of exogenously caused postural changes, to which we shall now make only a passing reference. In the literature, these residual conditions are undoubtedly considered to be related to postural changes in the vertebral column. Again Schmorl must top

our list; not only does he refer to secondary scoliosis (p. 233), but describes the mechanism by which associated disorders of the vertebrae and subjective complaints may arise (pp. 233, 216, Aufdermauer, Jenny). We shall deal with this fully in another chapter.

The angle between the sacrum and vertebral column may be larger or smaller than normal (Lewin, p. 497). Many authors regard this reduction in size as possibly the manifestation of a constitution predisposing the subject to low back pain (Goldwaith, Schmorl, p. 238). Exogenous factors, such as heavy manual labour for a prolonged period, are also thought to be partly responsible (Brailsford). It is not generally assumed, however, that there is a direct relation between any change that takes place in the angle between the sacrum and the spinal column and the occurrence of back trouble. Grimm, Herndon (quoted from Schmorl) and Robinson do believe that this relationship exists; Herndon noted a reduction of this angle in 68% of 498 cases of low back pain. We must put it on record that the reduction of this angle – *i.e.*, the closer approach, dorsally, between sacrum and vertebral column – occurs regularly with every exaggerated deepening of lumbar lordosis, as, for instance, during pregnancy. Hence it is by no means inconceivable that the primary cause of the back complaints in Herndon's material was an entirely different one, in association with which the lumbar lordosis became deeper secondarily and, consequently, the angle between the sacrum and vertebral column became smaller. Schmorl partly resolves this question, though he approaches it in a different way. He points out that the size of this angle depends on the shape of the fifth vertebral body and the condition of the intervertebral disc between L.V and S.I. The L.V vertebral body is wedge-shaped: in young people the difference in size between the anterior and posterior segments of this vertebra is 6 mm, while in older persons it is barely 7 mm (p. 237). These discrepancies, compared with the measurements recorded by other investigators, are too small to account for the change – in this case diminution – of the angle between the sacrum and the vertebral column. Far more stress is laid upon the L.V–S.I intervertebral disc, in which, after the 40th year, degeneration, tissue formation or shrinkage (in height) is presented in varying degrees.

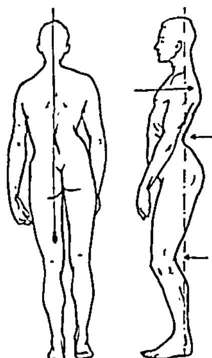


Hence in all probability the sagging of the spinal column or, as the case may be, tilting of the sacrum, can be equated with a change in posture; that is to say, a "disturbance" of the function of the joint between the spinal column and the sacrum. Seen in this light, the diminution or enlargement of the angle formed by the sacrum to the spinal column could be interpreted as a compensating mechanism in the assumption of the least painful posture. Accordingly, *changes in this angle* are in no way significant as a primary explanation of low back pain. They may occur:

endogeno-congenitally;
exogenously, *e.g.*, as the result of rickets and osteomalacia;
as a primary change of posture, *i.e.*, "slumping" when ligaments and muscles weaken;
as a secondary change of posture, *i.e.*, a reflex mechanism activated by the assumption of the least painful posture in the presence of a painful abnormality elsewhere in the body.

The correction of a lumbar lordosis as a *protective mechanism* against pain is to be regarded as one of the possible aspects of a mechanism of this kind. Generalising, we might say that the sensation of pain (in the back) impels the body to assume the posture in which the pain is least irksome. The choice of this least painful posture will depend upon whether the pain is caused by strain of the *static functions* or by *movement*. These two conditions will not always be separable, for the assumption of a painful posture need not necessarily involve movement of painful muscles or painful parts of the spinal column. On the other hand, any movement, whether painful or not, must affect the posture and if one particular posture is painful, the moving body will always take care not to assume it. Therefore, if pain-producing static factors are involved, the body will have to be on its guard against every movement as well as against a given posture, whereas with pain-producing movements, it will need only to avoid those movements. Relatively, therefore, pain resulting from strain of the static function of the vertebral column will evoke a protective mechanism oftener and more abruptly. The most effectual specific mechanism by which the static function of the vertebral column is relieved is undoubtedly the fixed flattened lumbar lordosis.

This reasoning receives support from the fact that a flattened lumbar lordosis is the postural change most frequently reported in the literature. Other mechanisms prevent recurrent small *movements* around the balanced position which are painful. This relieves the body of the necessity of assuming the most favourable posture relative to the strain on the body; all it need do is to *deflect the median line to the extreme position*, at which no further flexible movements take place around an equilibrium The greater the eccentricity, the more stable will



it be; and this ties up revealingly with the fact that the occurrence of a marked scoliosis is also reported to be associated with one-sided painful local disorders of the muscles (inflammation, abscess, tumour, trauma.)

The displacement of the median in the median line, *e.g.*, in the extreme position backwards, causing an exaggerated lumbar lordosis and, secondarily, diminution of the angle between sacrum and vertebral column, may be regarded as a simple mechanism for avoiding excessive movement and thereby, for avoiding painful changes of posture.

In conclusion we would touch briefly on the *coccyx*. Epstein, p. 116, Kinnier Wilson, p. 1433, Lewin, p. 550, report the total or partial aplasia of these vertebrae. This is said to be associated with many anomalies elsewhere, *viz.*, myelodysplasia, anomaly of the cauda equina, spina bifida, abnormalities of the urogenital system and of the pelvis. The authors do not mention complaints of pain. *Coccydynia* is an obscure syndrome on which opinions differ widely. Some physicians consider vague, nagging pain at the site of the coccyx to be entirely psychogenic in origin. Even the radiographic evidence of abnormalities, such as marked deviation of the angle relative to the sacrum and the confirmed existence of a trauma in the clinical history, cannot convince them of the organic source of these complaints (Lewin, p. 558). They maintain that the consistent failure of surgical intervention to relieve these complaints (extirpation of the coccyx) provides firm support for their contention. The temporary favourable response to infiltration of Novocaine between the coccyx and the sacrum, as well as the result of osteopathic manipulation (Zukschwerdt, p. 174), together with the radiographic evidence, constitute arguments in favour of the opposing opinion.

Although not strictly within the framework of this chapter, we would mention:

Spondylitis tuberculosa lumbalis is cited by several authors (Schmorl, p. 114) as one of the causes of low back pain. A monograph by Duggeli and Trendelenburg on this subject appeared as recently as 1957. Although the malady is exogenous, the patients' case histories often provide unmistakable evidence of an endogenous disposition to the infection.

The lumbar form, which often creeps on in its first manifestations, is very difficult to diagnose. It is when the disease is localised in this region that the latent period between infection and the clinical discovery of the focus is a long one, *viz.*, 12 to 30 months

In the case of chronic, unaccountable back trouble resisting all treatment, a case history showing a family taint or a tuberculous infection in the past certainly furnishes an indication for persistent, possibly clinical, examination. General symptoms, such as subfebrile temperature, lassitude and night-sweats, reinforce this indication. An increased sedimentation rate of the erythrocytes, discrepancies in the blood smear and positive Pirquet and Mantoux reactions call for radiological examination According to the aforementioned research workers, however, laboratory analysis fails to produce a single specific datum. *E.g.*, in a quarter to one-third of the cases the sedimentation rate is not increased; a considerable and constant rise would even provide an argument against a tuberculous process in the vertebral column.

Local tenderness would only occur in the case of an isolated process in the vertebral arch and processus spinosus or in that of a process in the iliosacral joints.

Radicular symptoms may result from cold abscesses, from local accumulations of tuberculous necrotic material around the foramina, or from the spreading of the process into the epimeningeal space. An active or healed process in the vertebral arch may induce spondylolisthesis, whereas the disintegration of a vertebral disc may lead to pseudo-spondylolisthesis.

It was understood that the intervertebral disc narrows because the tissue is attacked by the tuberculous process itself, either by way of a primary focus in the disc, or by invasion from a focus in the adjacent vertebral body. The authors we have just cited, however, assume that the contents of the intervertebral disc escape through perforations in the end plates to the cavity of the abscess in the adjacent tuberculous vertebra. Schmorl (pp. 116 and 199) mentions both possibilities.

Early diagnosis (first year) is also difficult to establish radiographically, even with the help of tomography; for the tuberculous process itself is not to be seen in the radiograph It is not until some time in the second year that the process becomes extensive enough (1 cm to 1.5 cm) to manifest itself by bone destruction and, sometimes, by reactive sclerosis in the surrounding bone. Schmorl (p. 115), on the other hand, puts the antecedent period at 3½ to 5 months. Before that time, however, an intervertebral disc involved in the process may have narrowed

Hence discal narrowing is an early symptom (first year) of vertebral tuberculosis, but appears to be by no means pathognomonic of the disease. Only

in children does the narrowing of an intervertebral disc point to the highly probable existence of spondylitis tuberculosa, since other forms of discal narrowing are so rare in early youth. The experienced radiologist has several criteria (such as, for example, local calcium impoverishment of the end plates) by which he can establish, with fair probability, the tuberculous aetiology of disc narrowing.

Diseases of the Bone Tissue

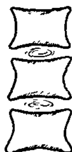
Osteoporosis

Osteoporosis is a deficiency of bone tissue as the result of an insufficiency of bone matrix (osteoid) with normal deposition of calcium. Put differently: the activity of the osteoblasts slows down, whereas that of the osteoclasts continues unabated.

Many skeletal diseases and various neurological and internal disorders are associated with a general osteoporosis, which is sometimes at its most pronounced in the vertebral bodies (including Cushing's syndrome, Basedow's disease, hyperthyroidism, Von Recklinghausen's disease as the result of hyperparathyroidism, ovarian agenesia). Scoliosis, kyphosis and all other resulting postural changes are caused secondarily. Mixed forms of exogenous and endogenous origin occur if the disease itself is caused endogenously. Osteoporosis appears fairly clearly on radiographs. The vertebrae change in shape, become narrower or wedge-shaped. These deformations may occur locally, as a rule at the place where the vertebrae are under most strain. Segments of the vertebra which have to bear most of the weight, *e.g.*, the joints, are apt to change first and become the source of spondylarthrosis. Accordingly, spondylosis and spondylarthrosis deformans are regarded as a reaction to osteoporosis of the vertebra, which attacks the frame progressively as the years advance. See also pre-senile osteoporosis, p. 83.

Osteomalacia (Schinz, p. 1046, Schmorl, p. 79, Groen)

This disease, which occurs only in adults, is a condition in which soft osteoid is formed instead of normal calcareous bone. It progresses intermittently, under the influence of endocrine disturbances, particularly those proceeding from the ovaries; accordingly, it is diagnosed most frequently in women at the end of pregnancy, accompanying menstrual disorganisation and after castration The worst affected in puerperal osteomalacia, so called, are the pelvis and sacrum The non-puerperal form – *i.e.*, that which is not associated with ovarian disorganisation – principally attacks the cervical, lowest thoracic, lumbar vertebrae and the sacrum It is as if the soft vertebral bodies sag under



any pressure exerted upon them; they become flatter and curve at the rims. At the site of the nucleus pulposus, which acts like a ball bearing, they appear to be hollowed out (fish vertebrae). The malady is recognizable, not only by the distinctive shape of the vertebrae, but also (radiographically) by the contrast between the decalcification of the spongiosa and the still calcareous cortex, which accentuates the resemblance to the vertebra of a fish. Snapper says that the normal vertebra in a radiograph appears as though painted with a thin brush, whereas the cortex of the osteomalacian vertebra exhibits the delicate structure of a pen-drawing. Hair-cracks are, moreover, to be seen here and there in the cortex of the bone and the discovery of these may help the physician to identify the disease in its incipience. Osteomalacia is characterised by atrophy of the bone and affects the patients with fractures, but chiefly by morphological changes of the parts of the skeleton affected owing to softness of the bone. The complaints come on gradually, being described as lassitude, discomfort in walking, pressure tenderness of the bones, pain in the lower part of the back and in the legs. In severe cases the patient has a slow, shuffling gait. The wobble of a sufferer from a congenital dislocation of the hip is reminiscent of the shambling gait which osteomalacia patients sometimes assume. The early detection of the signs of the disease in its incipient stages is important to prognosis and therapy. The latter consists in management with vitamins, calcium and hormone preparations together with radiation treatment. Secondary curvatures of the spinal column, pelvis and extremities set up a pathological motoricity. Any connection between this and subsequent trouble to which it may give rise will not appear from the literature, as it would be overlaid by the pain already caused by the idiopathic disease, itself.

In the clinic, the differential diagnosis of osteomalacia should include those pathological conditions which have been brought about by:

Chronic demineralisation of the skeleton

Upon microscopical examination, however, this is found to be associated with changes in the bony tissue strongly resembling the general osteoporosis attendant upon certain internal diseases. Epstein (p. 174) is of opinion that all forms of osteoporosis present the same image, both radiographically and microscopically. Spongiosa and cortex are linearly thin, yet consist of normally formed bony tissue. But the distance between the bony rods is abnormally large. The red bone-marrow proves to be largely replaced by fat tissue. In diagnosis, therefore, the cause of osteoporosis cannot be established on the

basis of pathological anatomy. Martin believes, however, that osteopathy due to disorganised metabolism is a type of osteoporosis with characteristics resembling osteomalacia (osteoid boundary). Its clinical diagnosis is difficult because the complaints are insidious. Even with severe demineralisation of the skeleton, the calcium content of the blood may remain normal (9.5 to 11.5 mg %), because it is constantly being supplemented with calcium from the bony skeleton. It is only with hypocalcaemia below 6 mg % that the concentration of Ca ions drops; hence conditions comparable to those associated with parathyroid tetany (hyper-reflexes, positive Chvosteck, etc.) will not guide us to the discovery of disturbed calcium metabolism. Nor should it be forgotten that osteoporosis is not always convincingly demonstrable by radiography. This needs subjective adjustment, being dependent upon the hardness or softness of the rays used. With a calcium shortage of 400 mg a day – a fairly high figure – only about 150 g are withdrawn from the calcium reserve per year, which is a loss that even the best radiographs cannot show (Martin). *A reduction in calcium reserve does not become radiographically demonstrable until it has reached 30% or thereabouts.* (More than 1000 g of calcium are combined with the bone tissue of an adult of 70 kg body weight, while only 10 g of this mineral are distributed elsewhere in the body.) As a rule, the cause of decalcification can only be traced by clinical investigation.

It is our impression that the chronic back pain from which some patients suffer is due to disorganised calcium metabolism. We find that they are generally middle-aged, with characters that might easily be mistaken for those of neurotics; they look old for their age, greying prematurely. If we say in this case that there is a question of constitution, we mean that these persons, on account of their disposition, are more likely than others to suffer the consequences of demineralisation, or else that, by virtue of their constitution, this demineralisation is more apt to take place. Epstein gives a list of the disorders that may possibly lead to chronic demineralisation of the skeleton, a list which we have supplemented with some points of our own:

Calcium deficiencies due to prolonged defective diet
Chronic loss of calcium through formation of fistula and drainage after gastro-intestinal operations
Sprue (in adults)
Chronic deficiency of Vitamin D and ultraviolet rays
Chronic overdosing of cortisone preparations
Relative hyperparathyroidism according to Snapper.
Pre-senile osteoporosis
Involutional osteoporosis



any pressure exerted upon them; they become flatter and curve at the rims. At the site of the nucleus pulposus, which acts like a ball bearing, they appear to be hollowed out (fish vertebrae) The malady is recognizable, not only by the distinctive shape of the vertebrae, but also (radiographically) by the contrast between the decalcification of the spongiosa and the still calcareous cortex, which accentuates the resemblance to the vertebra of a fish. Snapper says that the normal vertebra in a radiograph appears as though painted with a thin brush, whereas the cortex of the osteomalacian vertebra exhibits the delicate structure of a pen-drawing. Hair-cracks are, moreover, to be seen here and there in the cortex of the bone and the discovery of these may help the physician to idendify the disease in its incipience. Osteomalacia is characterised by atrophy of the bone and affects the patients with fractures, but chiefly by morphological changes of the parts of the skeleton affected owing to softness of the bone. The complaints come on gradually, being described as lassitude, discomfort in walking, pressure tenderness of the bones, pain in the lower part of the back and in the legs. In severe cases the patient has a slow, shuffling gait. The wobble of a sufferer from a congenital dislocation of the hip is reminiscent of the shambling gait which osteomalacia patients sometimes assume. The early detection of the signs of the disease in its incipient stages is important to prognosis and therapy. The latter consists in management with vitamins, calcium and hormone preparations together with radiation treatment. Secondary curvatures of the spinal column, pelvis and extremities set up a pathological motoricity. Any connection between this and subsequent trouble to which it may give rise will not appear from the literature, as it would be overlaid by the pain already caused by the idiopathic disease, itself.

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4. The clinical histories of these patients made it plain that, from their youth up, they had been accustomed to a diet deprived of the necessary amount of calcium.
5. The calcium balance of these patients was chronically negative.
(No other known causes of osteoporosis were found.)

The above authors reasoned as follows: Calcium disappears from the bones; it had been established that the loss of calcium per unit time was not above normal; hence there will be no more than the normal breakdown of bone. Seeing that for many years the patients' diet had included insufficient calcium, the ostensible calcium deficiency of the bone must be due to chronic insufficiency of calcium deposition while the breakdown of bone remained normal. In other words: the chronic loss of calcium is due to the fact that, in the physiological process of digestion of the bone tissue, the broken-down calcium is not completely replaced by the calcium in the food ingested.

The problem is therefore reduced to the following proposition:

Osteoporosis ((pre-)senile etc.) is brought on by a deficiency of calcium in the food.

subacutely: bone and calcium deficiencies may simulate starvation osteomalacia, but, with the simultaneous absence of other building bricks, *e.g.*, proteins, there will be an insufficiency of osteoid and, therefore, true starvation osteomalacia.

or chronically. through an insufficient intake of calcium (*e.g.*, through ceasing to take milk products after youth).
Only indigestible calcium was ingested (green vegetables).
Or else the re-absorption of calcium was disorganised (disordered digestion of fat).

Up to this point we can follow the authors' argument, particularly as, our attention having been drawn to it, we too were able to establish the fact that several patients suffering from osteoporosis, for which we were unable to account, had been accustomed to a diet poor in calcium since their youth. The problem raised by Groen and co-workers is very important because there are so many sufferers from undefinable and unknown back complaints. Are we, in fact, to consider an appreciable number of these complaints to be the result of (pre-)senile osteoporosis, or, put differently, are these persons suffering from an osteopathy resulting from chronic calcium deprivation or, as the case may be, a relative starvation osteomalacia? Groen suggests that the commencement of this osteoporosis – which he identifies, or at least compares, with (pre-)senile osteoporosis – depends on the extent of the calcium deficit since youth. Meulengracht is of the same opinion. Groen puts it like this "According to whether the calcium deficit in the food is small,

Although the results of creeping, chronic demineralisation do not altogether fit within the framework of this study, they are of such importance to the differential diagnosis of low back pain that they each deserve some comment.

Starvation osteomalacia (Groen and Merx, La Chapelle, Pompe) is distinguishable from osteomalacia only in its clinical history. It is caused by an insufficient supply of calcium and its incomplete re-absorption through the absence of fat from the food taken. Meanwhile, calcium is withdrawn from the skeleton to maintain the calcium level in the blood; calcium is passed through the bladder and elsewhere and eventually a negative calcium balance supervenes.

The starvation osteomalacia in Germany after World Wars I and II is described in the same way (Schmorl, p. 80).

The experiments performed by Brull on animals, which showed that, on the whole, the body maintains its normal chemical composition, even under the stress of extreme starvation, would seem to conflict with the view just expressed. Van Heusden, on the other hand, states that under starvation conditions the body does, it is true, hold on to the proteins, iron, sodium and potassium as long as possible, but excretes calcium and phosphates as usual. Considering that, where there is a very marked negative calcium balance (approx. 400 mg a day), it takes two years for the calcium reserves of the skeleton to be depleted sufficiently to be demonstrable by X-ray, one begins to wonder whether undue significance be not attached to calcium deficiency as the cause of starvation osteomalacia (Groen and Merx, p. 67, La Chapelle, Pompe). We suggest that a more probable cause is a combination of the simultaneous serious deficiency of protein, Vitamin D and, possibly, calcium, accentuated by a hormonal discrepancy (Van Heusden, p. 2504).

Calcium deficiencies due to prolonged defective diet. Groen, Duyvensz and Reidel noted, during their enquiry into the cause of parodontitis, that several patients suffering from it complained at the same time of pain in the back. After known organic maladies had been considered and eliminated, it was realised that the trouble was due to osteoporosis. When this was verified in a larger group of patients, this osteoporosis proved to be general, *i.e.*, over-all ostensible calcium deficiency of the skeleton (which in reality points to a deficiency of bone), more especially of the vertebral column and alveolar process. The disease was also found to run roughly parallel with age

We list the following facts extracted from the reports on this research:

1 These patients suffered from over-all osteoporosis.
2. The calcium content of the blood remained normal.
3 The quantity of calcium excreted in the urine and faeces continued to be normal to subnormal.

determine what quantitative deficiency is going to become pathological and when; *simultaneously*, the presumed hormonal disturbances will bring about those mental and physical changes which are undergone in the course of the disease. Seen in this way, the clinical pictures of involution and (pre-)senility remain distinct. At the same time, it can be seen why the symptoms occur under special circumstances, *e.g.*, castration, lactation, pregnancy and those conditions which adversely affect calcium metabolism, such as avitaminoses, deprivation of sunlight and the like.

There is good reason to believe that various circumstances exist which may predispose the subjects to the consequences of demineralisation, or may bring this about (avitaminosis, involution). *Each* of these factors, if strong enough, may be conducive to osteoporosis in a more or less specific guise. In the case of less compulsorily pathological factors a general cause will have to be far more pervasive. Seen in this light, the solution Groen suggests for this puzzling problem becomes easier to understand.

It is now also possible to see how it comes about that a few of those patients who have developed osteoporosis were found, on examining their nutritional history, to have been following a diet *rich* in calcium since their youth (*e.g.* a pint of milk a day)! For these hormonal disturbances in the metabolic process of the bone may well be rigorous enough to disorganise the absorption or deposition of calcium independently and need not necessarily be precipitated by an insufficiency of ingested calcium. It would be beyond the scope of our subject to pursue this matter further; our own purpose has been served by showing that osteoporosis, which may produce pain in the back, occurs as a symptom of what is probably an endogenous, hormonal, all-pervasive pathological change in man. We may add in passing that many authors, including Snapper, are of opinion that the metabolic process of the skeleton proceeds under hormonal influences. This does not only apply to adenoma of the glandulae parathyreoideae which Snapper describes in detail; in his description of bone physiology he constantly refers to the effect of the para-hormone of Collips. This is particularly clear where he states that hypertrophy of the accessory glands was observed as a secondary reaction to disorganised calcium metabolism (notably in association with osteoporosis).

The difficulties encountered in the theoretical and practical approach to this problem must be briefly touched on. The hormonal disturbances which lie at the foundation of this problem are not yet clearly understood. By "hormonal" we mean the influence exerted upon the metabolic process by which calcium is either not dissolved–broken down–re-absorbed, is not fixed in the body, or is deposited excessively into the tissues. The function of the sexual or other

considerable or very considerable, the disease does not occur at all, occurs *in old age* or *before old age*".

In our practice we encounter persons suffering from pain in the back (low lumbo-sacral or between the shoulders, etc.) and in whom osteoporosis is demonstrable in varying degrees of severity, for which the known causes, such as Cushing's disease, hyperthyroidism, disordered digestion of fat, etc., have, as in Groen's material, to be ruled out. We have also noticed that the patients are often middle-aged, usually women of more than 45. They look old for their age, go grey early or complain that their hair is falling out, by no means infrequently, these propensities run in families. On the whole, their blood pressure, far from being higher, is sometimes lower than that normally corresponding with their age. They are rather nervous and there are features of their character which are strongly reminiscent of neurotic traits. Disturbances definitely of a hormonal nature, which are also reported by Groen and his associates, often occur simultaneously, and twice as much in women as in men; *e.g.*, symptoms of involution, abrupt incidence or exacerbation of the symptoms after pregnancy or lactation, (pre-)senile lacunary dementia, etc. Avitaminosis C and D and lack of sunlight also appear to be factors, while the victims of rickets in youth seem to be predisposed to osteoporosis at a later age.

It is to be inferred from Groen's quantitative analysis that (pre-)senility, or involution, sets in once a calcium deficit exists of a certain number of grams (in ratio to the weight of the body). It would follow from this that (pre-)senility or involution could be prevented or deferred to a later period by an intensive consumption of calcium. And it would also follow that women reach this deficit of calcium earlier, or that, generally speaking, women, from adolescence onwards, eat food of lower calcium content. It would then have to be assumed that avitaminosis took place, a lactation period or a pregnancy had terminated at exactly the time when this calcium deficit was reached.

We ask ourselves whether the picture of the prematurely aged human being as outlined above should not be interpreted as a syndrome of hormonal disturbances, of which osteoporosis is little more than a symptom.

Together with avitaminoses, abnormal calcium drainage during lactation and pregnancy, in conjunction with disordered digestion of fat and demineralisation as the result of chronic gastric and intestinal fistulas, a diet chronically short of calcium is only a factor precipitating the disease. If the calcium deficit is very large, this deficiency must nesessarily become pathological and lead to a condition known in the literature as "starvation osteomalacia". If the chronic calcium deficit is small, the conditions prevailing in the body will

determine what quantitative deficiency is going to become pathological and when; *simultaneously*, the presumed hormonal disturbances will bring about those mental and physical changes which are undergone in the course of the disease. Seen in this way, the clinical pictures of involution and (pre-)senility remain distinct. At the same time, it can be seen why the symptoms occur under special circumstances, *e.g.*, castration, lactation, pregnancy and those conditions which adversely affect calcium metabolism, such as avitaminoses, deprivation of sunlight and the like.

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glands which influence the involution process begins to slow down gradually; thus a woman may complain of congestion and be swayed by the psyche of the climacteric for quite a time before menstruation shows signs of abatement.

We must repeat that osteoporosis can sometimes be observed in radiographs; but at first it is a subjective observation in the interpretation of which it is necessary to take into account the softness or hardness of the radiographs and also, say, the obesity of the patient. An extensive, often clinical, examination has to be carried out before other causes of an internal neurological nature, which are likewise liable to produce osteoporosis, can be ruled out. We are thinking of the possibility of disorganised fat metabolism, accelerated intestinal peristalsis and so on. The same suspicions are aroused if a negative calcium balance is found. Moreover, the calcium metabolism measured at examination is merely momentary, whereas it is precisely the *chronic* character of any disturbances there may be which is significant. According to Martin, a negative calcium balance can be tolerated for years without producing clinical symptoms or skeletal changes which are radiologically observable.

The medical practitioner is confronted with these complicated cases almost every day. Admirable though Groen's explanation be, both for its simplicity and the straightforwardness of the therapeutic management called for (by administering calcium), it would seem, in the light of these further considerations, inadequate for our complicated cases. Groen's discovery is important, because that rare condition, *viz.*, a subacute severe calcium deficiency, is pathological in a compulsory sense, also because a chronic slight calcium deficit could become pathological in the presence of factors which render the body sensitive to a deficiency of the kind. Nor does this explanation contradict the clinical view that, psychically, neurologically and endocrinologically, osteoporosis constitutes a symptomatic aspect of distinct, often endogenously produced clinical pictures. Within this context, osteoporosis falls into various categories, such as osteomalacian, symptomatic (faulty diet, avitaminosis, calcium drainage, etc.), post-climacteric involutional, (pre-)senile and probably many others.

Were we to try to represent *the foregoing schematically*, we should start from Groen's plan, thus·

Calcium disappears from the bones.

A negative calcium balance exists with normal excretion of calcium. The alternatives would then be:

I There is more than the normal breakdown of bone, though the deposition of calcium is normal (In that case the excretion of calcium should be above normal, which is contrary to the actual facts.)

II. The breakdown of bone is normal and there is reduced deposition of calcium.

III. The breakdown of bone is above normal and there is reduced deposition of calcium.

The conditions under II tally with the facts of the case and are used by Groen as the basis for his postulate that reduced deposition of calcium is due to a reduced supply of calcium.

The conditions under III, however, also fall into line with this postulate; for the increased breakdown of bone and the reduced deposition of calcium might be the result of disturbances in the metabolic process, *e.g.*, disturbances of a hormonal nature, in which case the supply of calcium plays a subsidiary part. Under certain circumstances the calcium balance will be negative, in equilibrium or even positive if the deposition has decreased less than the breakdown has increased. A fourth possibility then comes to mind, *viz.*,

IV. The breakdown of bone is a little below normal, while there is a more pronounced decline in calcium deposition.

Finally, there is a fifth point, when the calcium balance is mostly positive:

V. The breakdown of bone is normal, whereas the deposition of calcium has increased.

	Breakdown of bone	*Calcium deposition*	*Calcium supply*	*Excretion of calcium*	*Calcium balance*
	"Normal"				
1.	+ + +	- - -	> n + + + +	> n - - - -	E
2.			n + + +	n - - -	E
3.			< n + +	< n - -	E
	→ O			→ n - - -	→ -
	Re I				
4	+ + + +	- - -	> n + + + +	> n - - - - -	-
5.			n + + +	> n - - - -	-
6.			< n + +	n - - -	-
7			⩽ n +	< n - -	-
	→ O			→ n - - -	→ - -
	Re II				
8.	+ + +	- -	> n + + + +	> n - - - - -	-
9			n + + +	> n - - - -	-
10.			< n + +	n - - -	-
11.			⩽ n +	< n - -	-
	→ O			→ n - - -	→ - -

O = extra absorption of calcium *from* bone → osteoporosis
E = in equilibrium

	Breakdown of bone	*Calcium deposition*	*Calcium supply*	*Excretion of calcium*	*Calcium balance*
	Re III				
12.	++++	--	> n ++++	> n ------	--
13.			n +++	> n -----	--
14			< n ++	> n ----	--
15			≪ n +	n ---	--
16.			none	< n --	--
	→ O			→ n ---	→ ---
	Re IV				
17	++	-	> n ++++	> n -----	-
18.			n +++	> n ----	-
19			< n ++	n ---	-
20.			≪ n +	< n --	--
	→ O			→ n ---	→ --
	Re V				
21.	+++	----	> n ++++	n ---	+
22			n +++	< n --	+
	→ O			→ n ---	→ E
23.			> n ++	≪ n -	+
	→ OO			→ n ---	→ -
24			≪ n +	none	+
	→ OOO			→ n ---	→ --
25			*none*	*none*	
	→ OOOO			→ n ---	→ ---

O = extra absorption of calcium from bone → osteoporosis
E = in equilibrium

In this schema normal breakdown of bone is represented by +++ and normal deposition of calcium by --- To accord with this, the normal supply of calcium is represented by +++ and the normal excretion of calcium by ---. The disruptions of these equilibria are taken to be equal amounts

Taking 22 as an example, consider a normal supply of calcium (n +++) in conjunction with a normal breakdown of bone (+++) and increased deposition of calcium in the body (----), the excretion of calcium should then be less than normal (< n --), which would produce a positive calcium balance (+)

Since the body strives to maintain the calcium content of the blood at standard level, a normally functioning excretory system will try to evacuate a virtually normal quantity of calcium (< n -- → n---), the calcium balance thus coming into equilibrium (+ → E) This is effected by the body's extraction of an extra amount of calcium from the reserves in the bone (→ O).

The value of a schema of this kind may be seriously questioned. Yet on closer scrutiny it will be found to bring out some curious points. Hormonal disturbances in calcium metabolism (on the left) are compared side by side with varying conditions as far as calcium supply is concerned (in the centre). Let the supply of calcium drop far enough quantitatively and, in association with all forms of hormonal disturbances (on the left), conditions arise (on the right) which must inevitably lead to osteoporosis. This is supremely so when there is greater deposition than breakdown of calcium (re V). Even with a normal supply of calcium (22) the body must then absorb calcium from the calcium reserves in the bone in order to maintain the excretion of calcium and thereby the calcium content of the blood at standard level. Every decrease in calcium supply (23, 24, 25) necessitates greater absorption from the reserves.

With no supply of calcium, its absorption from bone is so intense that a condition arises such as prevails in starvation osteomalacia. The very valuable datum furnished by Groen's proposition is that a (chronically) reduced supply of calcium leads to osteoporosis; but we would add that the conditions – – e.g. created by hormones – under which this takes place vary considerably. If the endogenously (hormonally) determined calcium breakdown and deposition are not in equilibrium, any reduction in calcium supply will sooner or later bring about a calcium deficit, or a chronically negative calcium balance. The first section of the schema shows that an increase in calcium supply (1) is to no effect because these additional amounts are excreted again (Snapper). Calcium deposition and breakdown being in equilibrium, reduced calcium excretion should go along with reduced calcium supply. But, with a constant level of calcium in the blood (3) and a normal calcium excretory mechanism, the excretion of calcium per unit time remains constant (as proved by experiments performed by Groen and co-workers as well as others). The body has no alternative but to maintain the calcium level in the blood by breaking down more bone (→ O). In that case the excretion of calcium is normal with reduced calcium supply and this is tantamount to extra breaking down of bone, *i.e.*, *osteoporosis*.

These mechanisms recur in the schema (3, 7, 11, 16, 20, 22, 24, 25). Irrespective of the hormonal conditions, whenever the supply of calcium is so small that the calcium level in the blood threatens to drop below normal on account of persistent excretion of a constant amount of calcium, more calcium is broken down from bone, leading inevitably to an extra negative calcium balance and osteoporosis.

The fifth section of the schema is particularly instructive. The deposition of calcium in the body is progressive from the 20th year of human life and

may be regarded as physiological. On the clear evidence of the schema, the calcium balance would become positive if the supply of calcium (21) were intensified (> n). This would offer the body a dangerous inducement to resort to more than physiological calcium deposition, *e.g.*, in the joints, ligaments, the connective tissue and the arteries, etc. The body wants to get rid of this calcium. This poses the question as to whether this economy in calcium supply, or consumption, be not physiological; in other words, whether people of advancing age do not instinctively avoid partaking of food that is rich in calcium

The following would seem to us to be an acceptable *hypothesis*. In youth the body uses up much calcium for the construction of the skeleton. Calcium is again released, admittedly, in the process of bone metabolism, but the calcium balance remains positive (more absorbed than given off). If this is not sufficiently positive, then, whether Vitamin D, etc. be taken or not, rachitic symptoms are presented. The work of building up the skeleton is complete by the time the body is adult; skeletal reconstruction still requires an intensive metabolic process; the arrest of growth is determined not only by the thickness of the epiphysial lines, but also by hormonal action, in which there is a gradual transition. From the 20th year a surplus of calcium begins to settle out in the body. If it is correct to suppose that man and beast instinctively choose the food their bodies most require, the mature human being will keep his metabolism in balance by limiting his intake of chalky food once the construction of the skeleton has been completed.

We are aware that some doubt exists as to the correctness of this assumption. Experiments on animals have shown that there is no sure evidence of the existence of an instinctive choice of food in animals removed from their natural surroundings. Animals living in the wild state are, however, assumed to possess that instinct. Man, whose purely instinctive impulses are largely submerged in the dominating functions of reason, is not thought to choose his food primarily by instinct; his choice of food is governed far more by social, cultural and traditional imperatives (Stemmer). For all that, it is noteworthy that, as far as we have been able to ascertain roughly, the most usual dishes with a high calcium content – notably milk products – tend to vanish more and more from the adult's menu. It is, of course, true that people also sometimes eat things that are not good for them. Sufferers from migraine, for instance, not infrequently have a preference for foods largely made up of purine derivatives.

An enquiry pursued by Lyons and Trulson brought some interesting facts to light. They questioned 100 inhabitants, aged between 65 and 80, of one of the poorer quarters of Boston about their customary fare. Nearly a quarter of

them consumed less than 75% of the quantities of calories, *calcium*, iron, thiamine, riboflavin and ascorbic acid recommended by the National Research Council. Among the foodstuffs lacking in the majority of cases were *milk*, cheese and green vegetables. For all that, 48% of the men and 57% of the women in the group examined weighed 10% or more above the average. No relation could be found between the degree of surplus weight and the intake of calories. Jansen (p. 312) also writes about the instinctive choice of food in man.

The foregoing would seem to show that the aged are physiologically impelled to restrict their consumption of certain elementary constructive units for the body, without thereby greatly impairing health.

Under hormonal influences, the metabolic process unmistakably begins to decline in intensity from maturity to senility. Fewer and fewer amounts of calcium will be liberated per unit time and thus a smaller absorption of calcium will be capable of compensating for this loss. (We are here ignoring the fact that the liberated minerals are in part utilised elsewhere for the bone metabolism.) To our mind, a declining absorption of calcium in late maturity is physiological; if it were to remain quantitatively the same as during the build-up period of the skeleton, either calcium diuresis would have to increase, or a dangerous surplus of calcium would be piling up in the body. One of the dangers of increased calcium diuresis is the formation of calculi and anuria (blockage of the tubules). Therefore *the absorption of less calcium* is a protective mechanism available to the body.

We cannot help asking ourselves, therefore, whether calcium therapy has any great value in the majority of these cases. Either it will be *useless*, because the calcium is in any event excreted again (Snapper), or *dangerous*, as this extra calcium is deposited in the body. Groen's proposition retains its validity, because the supply of calcium should not fall below a minimum. The actual therapy should still be aimed at rectifying the disorganisation of the process of calcium breakdown and calcium deposition. It is for this latter reason that we have dwelt at length upon this matter, because, when confronted with patients complaining of back trouble which proves to be the result of osteoporosis, we should be able to differentiate between the various clinical pictures in order to adopt effective therapeutic management.

The *literature* provides support for the views set forth above. Epstein, Lewin and many others state that osteoporosis results from an imbalance between calcium breakdown and calcium deposition. This imbalance is said to be hormonal in origin (Albright, Cocchi, Flink – quoted by Epstein – Fraser, Michelson, Radwin, Reifenstein, Richardson, Shore, Smith, Snapper). For the

therapeutic management of pre-senile osteoporosis, Freyberg and Levy advise warning patients passing through the post-climacteric period against consuming large amounts of protein, calcium and phosphates.

Two facts established by experience form the background of our schema, *viz.*, the constancy of the percentage of calcium in the blood serum (Albright, Epstein, Snapper) and the passing of a normal quantity of calcium in the urine per unit time (Albright, Epstein, Groen, Reifenstein). This is again confirmed in the records of recent research done by Gerbrandy and Ebelink; they found an almost constant percentage of calcium in the blood serum of patients suffering from osteoplastic and osteolytic tumours of the bone. There existed an exceedingly significant correlation between the passing of calcium in the urine and the calcium level in the blood. As was to be expected, the patients with predominantly osteoplastic skeletal abnormalities passed little calcium, whereas the patients with osteolytic skeletal anomalies in large measure had calciuria. This increased excretion of calcium ran parallel to the extent of the radiologically demonstrable osteolytic abnormalities. This difference in calcium excretion was manifest, although both groups were on a diet poor in calcium.

In conclusion we quote Snapper (p. 4), who says, "The higher the calcium content of the blood serum, the larger the amount of calcium excreted", and elsewhere, "The supply of large amounts of calcium is to no avail, as it is passed out of the body again in any event".

Chronic loss of calcium resulting from fistula formation with visceral short-circuiting and from prolonged drainage after gastro-intestinal operations

The absorption of calcium takes place in the small intestine. Apart from being passed through the kidneys, calcium is excreted in the large intestine, where no calcium is re-absorbed. If the small intestine is not functioning, yet calcium is lost *via* urine and faeces, the result may be a deficiency of calcium. *Although the food contains sufficiently digestible calcium, the body does not* benefit from it.

This is what happens when fistulas have been formed between the stomach, duodenum and colon, the larger the section of the duodenum short-circuited, the more likely is the absorption of calcium to become disorganised.

After a gastric resection, only small parts of the duodenum are put out of action, where there is an anus praeternaturalis the major part of the ileum continues to function, but the passage of food is irregular; the danger of insufficient calcium absorption certainly exists, especially if the disorder is of long duration. Experiments on animals (Loewy, quoted by Snapper, Part I)

have shown that osteoporosis sets in after only a few weeks, spontaneous fractures occurring within two months. Osteoporosis is known to be liable to occur in association with ileitis terminalis when fistulas have been formed between high loops in the small intestine and the colon (Prick, Snapper). There can be no doubt that the inadequate absorption of calcium which it involves is partly due to the impaired functioning of the changed mucosa. We get the same thing in the case of accelerated peristalsis to such a degree that the duodenum is given no opportunity to cope with the food, more specifically to absorb calcium. As far as we are aware, it is not yet known whether osteoporosis as the result of hyperthyroidism is caused by endocrine changes only, by hyperperistalsis or by a combination of the two.

Osteoporosis as the result of disorganised absorption of fatty acids and soaps

The following applies to some extent to the pathological conditions involving disorganisation of the digestion of fats. The small gut can only extract calcium from food if this is present in an ionised form in the intestinal canal. Vitamin D is responsible for the passage of ionised calcium through the intestinal wall; this vitamin has to be liberated by pancreatic and intestinal lipases from the fat in which it is dissolved. Even under normal circumstances the almost neutral reaction in the duodenum leaves the calcium free to form insoluble compounds. When the process of fat absorption is disturbed, large amounts of free fatty acids are left behind in the duodenum and these may combine with so much calcium that absorption of calcium begins to fail and, consequently, the skeleton becomes impoverished (Groen).

Disorganisation of fat absorption may take place as follows:

1. The fats have to be saponified to produce free fatty acids and soaps. Before these can be absorbed by the intestinal wall, they have to be emulsified by the bile. If there is insufficient secretion of bile due to a diseased liver or blockage of the bile ducts, the fatty acids and soaps are not absorbed and remain in the small intestine.
2. The intestinal mucosa must be normal and the chylous vessels must function properly if the absorption of the fatty acids and soaps is to proceed in the way it should. Under certain conditions, such as atrophy of the mucosa, tuberculosis and the growth of tumours with closure of the efferent ducts, the mucosa and chylous vessels are not capable of absorbing fatty acids and soaps.
3. Visceral short-circuiting may deprive the gut of the ability to absorb food, hence not only the calcium present, but also the broken-down fatty substances.
4. If peristalsis is chronically accelerated, the food may pass too quickly for the gut to be able to absorb the digested products of the ingested food. Thus undigested fat is known to be in the faeces excreted by patients suffering from Basedow's disease.

This type of osteoporosis is described in the literature by Cocchi, Epstein,

Groen, Snapper and others. Meulengracht ascribed involutional osteoporosis to it.

The most familiar causes are destruction of the hepatic parenchyme (cirrhosis, metastases), tumours in the abdominal gland packets, closure of the bile ducts and gall-bladder fistulas outwards. Epstein says that Snapper and others had noticed that women whose hormonal activity was already disturbed are prone to this osteoporosis resulting from disorganised fat metabolism.

We wish particularly to emphasize *sprue*, this being a less well-known clinical picture and, as we have found, being associated with back complaints due to osteoporosis. The duodenal mucosa of adults suffering from sprue is diseased and atrophic. The free fatty acids remain largely unabsorbed for periods of varying length in the intestinal tract. The patient is intermittently constipated; then, suddenly, there comes a brief period of diarrhoea with fat in the stools, which are putty-coloured, frothy and acid. With violent attacks of this nature, the patient will soon be under specialist treatment for chronic diarrhoea. These explosive attacks of bulky fat diarrhoea are characteristic of the chronic and often mistaken forms of sprue. The patients complain of itching, they become emaciated, the mucous membranes are pigmented. Hyperchromic anaemia often supervenes, with elliptical megalocytes in the blood. The internal secretion becomes disorganised, *viz.*, basal metabolism increases and the blood pressure is lowered; loss of strength and tetany have been described as being associated with the condition. Osteoporosis is very liable to occur, with tenderness of the bones and back trouble. Osteomalacia seldom develops.

Chronic deficiency of Vitamin D and ultraviolet rays

Far more is needed for the normal build-up and maintenance of the skeleton than merely absorbable calcium. If, for instance, there should be any interference with the protein metabolism, as in Cushing's disease, the result will be osteoporosis. Chief among the known building bricks are calcium and proteins, while the presence of hormones, enzymes (osteoclastic, osteoblastic). Vitamins D, C (and A?) is needed for metabolism to proceed in the bone.

Ultraviolet rays on the skin (or on certain food) can activate the sterol it contains to ergosterol, thereby supplying the body with a natural source of a substance having the effect of Vitamin D, able to replace Vitamin D and supplement any deficiency of it. If the food given to children contains the requisite quantity of Vitamin D, the absence of light will not cause them to become rachitic (Lewin, p 459)

It came to be realised that an absolute deficiency of one of the above sub-

stances is bound to be pathological for the metabolic process of the skeleton. There are, however, any number of possible combinations of deficiencies which, in a given constellation, might lead to osteoporosis. It is held that, faced with a relative deficiency of one of these building bricks, the body will find a way to make up for this lack; but, if several of the circumstances are adverse, presumably a pathological condition will be created sooner if there is a considerable deficiency of one of the other substances mentioned.

Thus conditions do arise which seem to point to the fact that building bricks plus vitamins are not enough for human beings of advancing age.

Complaints of pain in the back and radicular pain are made more frequently by nuns of middle age than among the average lay population, without evidence that a disc lesion is primarily responsible for these complaints; but, upon exploration, it was found that the bony part of the vertebral column was softer than is normally the case. We noticed on more than one occasion that the arches were distorted in the vertebral canal and that the foramina had narrowed to a flat oval. Osteoporosis in a mild degree was usually demonstrable in the radiographs. Anatomical features other than those known to be associated with a mild form of osteoporosis were not found in the removed pieces of bone by the pathologist. This softness of the skeleton caused progressive deformity of the vertebral column in several cases sooner or later after laminectomy.

By virtue of what conditions does this group of people stand out from the average population? The only significant difference in the mode of life we can find is the fact that the nuns spend almost the whole of their time indoors and, when they do emerge into the open air, their bodies are almost completely insulated by the thick, voluminous habit, while even their faces are shaded by the projecting hood. In so far as we were able to ascertain, there were no other conditions markedly different from those under which the average population lives. Thus the diet was certainly not chronically poor in calcium and proteins, nor of such calibre as to make us suspect a Vitamin C or D deficiency.

Apart from the operative observations mentioned, the incidence of this osteomalacian-osteoporotic condition was reported simultaneously in the Netherlands in the post-war years (1945, 1946, 1947) by Groen, De Jong, Pompen, Prick and co-workers (p. 65). At the time it was ascribed to the prolonged undernourishment suffered by the people. We know it to be a fact that at that time more nuns were affected by this condition than nowadays under normal circumstances. When we now consider that osteoporosis may be due to a variety of causes, notably a deficiency of calcium, proteins, Vitamin D, a hormonal deficit and lack of ultraviolet rays, it may be surmised that nuns,

who, on account of their clothing, receive insufficient benefit from ultraviolet rays, are more prone to osteoporosis, especially if other pathological conditions produced by the pathogenic factors mentioned are already present. This view would seem to be strengthened by the greater incidence of the disease in the climacteric and its incidence among the younger nuns after a long period of near-starvation.

If the above observations represent the true facts, they suggest very tentatively that middle-aged women, *i.e.*, at or near the climacteric, need more than an otherwise sufficient dose of Vitamin D to maintain the metabolism of their skeleton. The question now arises whether, through exposure to ultraviolet rays, substances are formed in the skin which can*not* be replaced by the Vitamin D in food. If so, these substances would be indispensable for normal metabolism of the skeleton when the hormonal functions begin to wane in middle age. Vitamin D without light, especially sunlight, prevents rickets at an early age. Ultraviolet light without Vitamin D likewise prevents rickets. Vitamin D and ergosterol generated by solar rays can replace each other.

Thus middle-age ailment due to lack of light is not prevented by Vitamin D; therefore, this disease of the bone is not "the adult's rickets", as, if it were, Vitamin D *would* compensate for the lack of ergosterol.

Is it the case that Vitamin D alone cannot cope with an endocrinological discrepancy, so that a rachitic picture is presented? Does light normally activate other substances besides ergosterol in the human skin, the lack of which is not felt until the internal secretory glands begin to fail?

There are reasons, we believe, for not ruling out the possibility that the action of ultraviolet rays on the skin stimulates other biochemical mechanisms necessary to bone metabolism beyond the known balance between Vitamin D and ergosterol.

Chronic (over-)dosing with Cortisone preparations

A patient aged about 50, who for years had been suffering from a rheumatic complaint, lifted an object of no great weight and suddenly felt a pain in his back radiating violently to the right leg This pain had a monoradicular distribution There was no evidence of a disc lesion Pronounced osteoporosis of the whole skeleton, but particularly of the lumbo-sacral vertebral column, was visible on the radiograph; the [illegible]

nothing abnormal had been detected in the spinal column, notably no marked decalcification.

The risk of skeletal decalcification through the prolonged use of Cortisone is

known in the literature. Epstein (p. 176) adduces evidence to that effect; he mentions spontaneous fractures and adds that nearly all these patients were being treated for rheumatoid ailments. Considerable loss of nitrogen is



incurred by overdoses of Cortisone and this leads to a shortage of proteins as building bricks for the bone matrix (osteoid). At the same time more calcium is lost through increased excretion and less absorption in the gut (Albright).

Epstein (p. 174) points out that there were predisposing factors in all these patients, such as advanced age, prolonged confinement to bed and the attainment of the menopause. He offers a complicated explanation of the katabolic and anabolic influence of the sex hormones on the metabolism of the skeleton and attributes an anti-anabolic action to the adrenocorticotropic hormones.

Within the framework of this study, it is important to realise that people exist whose skeleton decalcifies at a certain period – probably under the influence of endogenously determined factors – as the result of taking Cortisone in doses that do *not* produce decalcification in other people.

Relative hyperparathyroidism

A distinction has to be made between adenoma of the accessory glands, which is the *cause* of Von Recklinghausen's disease, and hyperplasia of these glands, which may be the *result* – or, more prudently, an accompanying symptom – of

skeletal decalcification. (Christopher, p. 299, Epstein, p. 176, with mention of several other authors, and Snapper, Part. I, p. 80.) Snapper deals with this subject in full detail and records the post-mortem findings and at operation in cases of pronounced skeletal decalcification; also the result of experiments on animals. He sums up as follows:

"Adenoma of the accessory gland plays a primary part in Von Recklinghausen's disease; this tumour *causes* disease of the bone. Other diseases, besides Von Recklinghausen's, are found associated with decalcification of the skeleton, in which hyperplasia of the accessory glands develops *as the result* of disorganisation of calcium metabolism (Erdheim, quoted by Snapper).

It has been found possible to induce this hyperplasia of the accessory glands experimentally by:

a Depriving animals, especially chickens, of Vitamin D or ultraviolet rays.
b. Continual withdrawal of bile from humans and mammals.

In clinical practice this secondary hyperplasia of the accessory glands should be borne in mind when confronted with:

a Osteomalacia and rickets.
b Chronic renal insufficiency, particularly in children (renal rachitis).
c. Exceptional cases of multiple myeloma and carcinomatosis of the skeleton.

It is well to remember that these maladies do not as a rule occur in conjunction with hyperplasia of the accessory glands. Only in rare cases has it been found at autopsy."

We would further quote Snapper as stating that he considers this hyperplasia to be occupational, in holding which view he relies, among other things, on the conclusion drawn by Loewy who, after experiments on animals, inferred that the decalcification of the skeleton resulting from disturbed fat metabolism proceeds along the following lines:

1 Loss of calcium with fat in the faeces
2 Diminution of the ionised calcium of the blood
3 *As a result, stimulation of the accessory glands, this leading to hyperplasia.*
4. Decalcification of the skeleton due to increased parahormone excretion.
5. Consequent supplementation of the calcium content of the blood up to the normal level

It is evident from this conception that it is not yet clear whether this hyperplasia is to be regarded as an *accompanying symptom* of pronounced osteoporosis,

or the indirect *cause*, while it appears from the above schema that it is not the *result* of decalcification.

This uncertainty is pertinent to our enquiry only in that it implies, with a high degree of probability, that several factors come into the picture.

It is emphatically stated that hyperplasia of the accessory gland does not *as a rule* occur in conjunction with marked osteoporosis. This argues directly against the assumption of a quantitatively absolute causal relation between the degree of the osteoporosis and the amount of a calcium deficit, or the seriousness of a chronic dietary defect.

In a later publication (1953) Snapper states that the patients who developed a disease of the bone through disorganised fat metabolism were old women, in whose cases the possibility of a senile or menopause osteoporosis could not be ruled out.

Under other conditions Hitzig, Albright and Reifenstein likewise hint at predisposing hormonal influences bearing upon the development of osteoporosis.

We have to ask ourselves whether hyperplasia of the accessory glands, which apparently occurs seldom under the same circumstances, is the resultant of several factors.

It follows from our survey of the literature that several authors credit hormonal influences, age, climacteric, involution, etc. with playing a large part in this matter. In the practical treatment for low back pain the demonstrability of hyperplastic accessory glands at autopsy in rare cases is of no significance; certainly not since it has been found that the results of removing one or two normal accessory glands in cases of ankylosing polyarthritis, etc. are, to say the least, still uncertain (Snapper).

Our purpose in lightly touching on these problems was to clarify what follows

Pre-senile osteoporosis – involutional osteoporosis

We must recall a few established facts before we attempt to describe these clinical pictures, *viz.*,

1. We do not yet know what makes human beings "grow old".
2. The processes of wear and tear are microscopically and biochemically demonstrable, but they cannot be physiologically explained.
3. Some people age prematurely, both mentally and physically.
4. Pathological processes and wear and tear occur in some people with considerable frequency, in step with symptoms of involution.
5. These involutional symptoms may be premature.

skeletal decalcification. (Christopher, p. 299, Epstein, p. 176, with mention of several other authors, and Snapper, Part. I, p. 80.) Snapper deals with this subject in full detail and records the post-mortem findings and at operation in cases of pronounced skeletal decalcification; also the result of experiments on animals. He sums up as follows:

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5. Consequent supplementation of the calcium content of the blood up to the normal level

It is evident from this conception that it is not yet clear whether this hyperplasia is to be regarded as an *accompanying symptom* of pronounced osteoporosis,

and the still incomplete *adaptation* to it by the remainder of the body and the psyche. The disease overtakes the patient in a period of life when he is still planning eagerly for the future; he may just have been promoted to a post demanding the best he has to give, it is just at this period that the greatest strain is put upon a woman by her growing family; women especially resent and resist the insidious encroachments of age and defy them by undiminished activity. Moreover, most people are still young enough in heart, clear-headed and muscular to do things which are getting beyond the tolerance of other parts of their bodies; this applies more especially to the supporting frame, which has to bear relatively too much strain.

Involutional osteoporosis (Jesserer) is called by others "the osteoporosis of the climacteric, post-climacteric and post-menopause" (Chaouah, De Snoo, Sleeswijk, Snapper, Van Assen). We must begin by stating that we have the impression that these and many other authors consider the causal relation between the occurrence of osteoporosis and low back pain, on the one hand, and a hormonal discrepancy during involution, on the other, as having been established.

Epstein's opinion is that osteoporosis is predominantly a disease of maturity and that there is a deficiency of oestrogenous hormones in the post-menopause, so that there is therefore no normal osteoblast activity.

Involution is understood to be a *complex* of physical and mental symptoms due, it is thought, to reduced activity of hormonal glandular tissue, especially that of the gonads. This impaired function, disorganisation or relative discrepancy, as the case may be, will have long preceded its outward manifestations as *e.g.* irregular menstrual periods. Now it is a fact of experience that patients of a certain category come to consult us, complaining in vaguely defined terms about their backs, all of whom could be described as being, roughly speaking, of the same constitutional type. They are usually middle-aged women – and there are also some elderly men – looking old for their age. They go grey early and this seems to run in families. Their character is of a kind very reminiscent of that of neurotics, with a depressive, moody undercurrent. Quite often there is incipient corpulence, their teeth are bad, or have been repeatedly treated, or else have been completely replaced by dentures.

Few neurological abnormalities are found; usually some tenderness is felt upon pressure in the superficial parts of the skeleton (pelvic crest, symphysis, tibia and also the base of the vertebral column). Quite often they complain of signs of change, particularly of diminished libido.

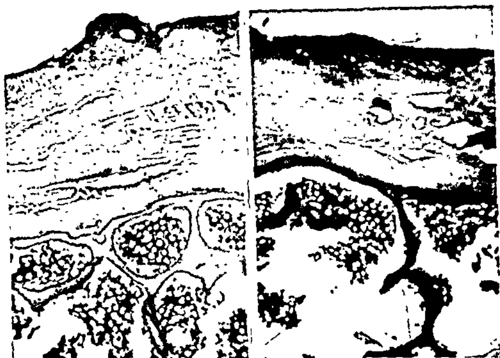
Approximately the same type of constitution is described in more general terms as belonging to individuals predisposed to demineralisation of the

By "premature" we mean that in the pre-senile period a condition prevails which rightly belongs to the senile period and that symptoms of involution are presented before the age at which these customarily occur.

The transitions are so gradual and vary so much from individual to individual, that we cannot tie them down to any particular age, such as the 65th or 50th year (Burger, De Snoo, Epstein, Fleischer, Jesserer, Schinz, Schmorl, Sleeswijk, Snapper, Todd and many others). Then, this premature ageing is seen to occur without any demonstrable external cause, such as leading a life making exacting demands upon the person's vitality. The incidence of both involutional and (pre-)senile osteoporosis in women is approximately five times that in men (Jesserer).

As both conditions undoubtedly occur in families, one might be tempted to consider the degree in which and the period at which they are presented as endogenously hereditary in families. When pre-senility and involution begin several years earlier than is to be expected, they might be called a premature physiological process. The phenomenon becomes pathological when severe symptoms are precocious and particularly when the individual is neither mentally nor physically ready to cope with this premature ageing. This will certainly stand out clearly if senility – possibly on a constitutional basis – is attained early as the result of mental and physical strain, or when an abrupt disturbance of hormonal relations in young people brings about symptoms of involution. It is incorrect to imply that the involutional syndrome, as far as osteoporosis is concerned, proceeds gradually from the pre-senile to the senile period, because both the syndrome itself and the complaints resulting from the accompanying osteoporosis may be entirely different.

Microscopically, *pre-senile osteoporosis* is indistinguishable from other forms of osteoporosis. If the decalcification of the bone is not greater than what is appropriate to the pre-senile period, there is no gradual transition to senile osteoporosis, which is irreparable and progressive: it causes deformities which are not generally painful (Schmorl, p. 76). If there is a degree of decalcification in pre-senility such as is not usually encountered until senility has set in, malformations will occur at the same time. The fact that these same deformities which are already present in the pre-senile age *are* painful, is an interesting functional phenomenon. Actually it is not surprising if those same pathological conditions which are borne with resignation by a patient passing through a quiescent age should cause distress when they assail a person in a vital period of life. ("Vitalitatsverlust", Albright.) Indeed, the acute painfulness of the pathological form of pre-senile osteoporosis is directly traceable to the incongruity between the premature ageing of certain parts of the body



Slight osteoporosis, thin cortex, thin bone trabecula covered with a very thick layer (painful on pressing?) of connective tissue containing very few cells.

pain does not occur until the disease has become severe enough for local changes to have attacked *adjacent* structures, *e.g.*, the periosteum. There are strong arguments in favour of this hypothesis in view of the following. The onset of the condition is subacute. After a period in which the patient suffers great pain and which may last for several years, the complaints diminish considerably or may clear up altogether, though atrophy of the bone continues to be visible radiographically. This indicates some measure of adaptation which the relaxation of the climacterial mental strains and stresses could not alone accomplish. The healing of secondary lesions (such as displacement or inflammation of the periosteum) would seem a more likely explanation. Serious secondary reactions, like morphological changes of the vertebrae and arthrosis, do, of course, persist and may continue to cause distress. Accordingly, the object of therapeutic treatment, *e.g.*, in the form of hormone substitution preparations, should be to prevent these secondary changes from taking place.

At an advanced stage of the disease an osteoporotic vertebra may collapse (spontaneous impression fracture); this may be accompanied by violent

skeleton, and, elsewhere, as associated with arthrosis deformans. We are not surprised that roughly the same description will apply presently to a group of persons more than normally distressed by an ostensibly normal motility.

Involution may be described as a physiological process. Endogenous factors determine the severity of the accompanying symptoms, the period at which it manifests itself and the duration of the life period in which this process of transformation is consummated. Upon those factors will also depend whether and in what degree decalcification of the skeleton takes place.

The most severe symptoms arise when the hormonal balance is suddenly upset in an unphysiological manner, *e.g.*, by castration. Even then, however, we do not know why decalcification of the skeleton does take place in one case and not to any great extent in another, or is sometimes deferred until long after the castration. If a hormonal revolution of this magnitude is imposed upon a young body that has not had time to prepare itself gradually for the coming change, the psychic components of the syndrome are apt to predominate. Finally, the (unphysiological) onset of the involution syndrome may be *very premature*, probably induced by endogenous factors. For this reason, too, concomitant osteoporosis, if present, is likewise determined constitutionally in respect of its severity and the period at which it manifests itself. Under the influence of endocrine disturbances, general atrophy of the bones sets in, attacking more particularly the vertebral column and pelvis, in contradistinction to senile osteoporosis, in which the whole skeleton gradually decalcifies (Jesserer). The conditions under which the disease occurs seem similar to those of osteomalacia Care should be taken not to confuse involutional osteoporosis with an undeveloped form of osteomalacia; which, though barely distinguishable from the former in radiographs, nevertheless reacts far better to hormonal therapy Every woman in the climacteric bearing signs of premature old age and complaining of pain in the back, loins, pelvis, shoulders, neck, and tenderness of the bones should be suspected of having the disease. Sometimes it becomes very painful as it progresses; it develops in a short time, usually after cessation of the ovarian functions at an early age.

Constitutional factors are certainly involved, because not all women experiencing the above come to suffer from the disease. (Literature: Albright, De Snoo, Jesserer, Kienbock, Schinz, p. 1134, Schmorl, p. 76, Sleeswijk II, p. 607.) On the causal mechanism of the pain the literature gives no views other than those expressed in relation to other malformations, dysfunctions, strain and secondary changes.

It is a known fact that atrophy of the bone can cause pain on pressure, but also spontaneously. It is not stated how this can be, nor whether such local



Slight osteoporosis, thin cortex, thin bone trabecula covered with a very thick layer (painful on pressing?) of connective tissue containing very few cells.

pain does not occur until the disease has become severe enough for local changes to have attacked *adjacent* structures, *e.g.*, the periosteum. There are strong arguments in favour of this hypothesis in view of the following. The onset of the condition is subacute. After a period in which the patient suffers great pain and which may last for several years, the complaints diminish considerably or may clear up altogether, though atrophy of the bone continues to be visible radiographically. This indicates some measure of adaptation which the relaxation of the climacterial mental strains and stresses could not alone accomplish. The healing of secondary lesions (such as displacement or inflammation of the periosteum) would seem a more likely explanation. Serious secondary reactions, like morphological changes of the vertebrae and arthrosis, do, of course, persist and may continue to cause distress. Accordingly, the object of therapeutic treatment, *e.g.*, in the form of hormone substitution preparations, should be to prevent these secondary changes from taking place.

At an advanced stage of the disease an osteoporotic vertebra may collapse (spontaneous impression fracture); this may be accompanied by violent

attacks of pain, which are capable of simulating a heart infarct (Jesserer) and result in considerable shortening of body length.

We come to the following *antithesis*: Pre-senile osteoporosis could be described as a physiological – or, in its severity and time of incidence, pathological – preliminary stage of senile osteoporosis, the latter being irreparable and progressive, qualifying only for palliative treatment. Involutional osteoporosis may present the same changes to the microscopist, but, from the very nature of its aetiology, it is preventable and, in the early stage, can be combated by substitutional therapy. It is of the first importance that involutional osteoporosis should be recognised as a clinical picture unto itself in good time so that appropriate steps can be taken to prevent secondary irreparable changes from taking place.

Jesserer and Kirchmayer (1956) summarise involutional osteoporoses succinctly but with admirable clarity. Their classification varies from that adopted in the literature as far as we are aware.

	Age						
	45	50	55	60	65	70	75
Jesserer.		*Pre-senile* involutional osteoporosis.			*Senile* involutional osteoporosis		
Literature	Involutional osteoporosis.			Pre-senile osteoporosis			
					Senile osteoporosis		

The latter conception seems to us the more correct. Senile osteoporosis is regarded as a "physiological" process which becomes pathological if it takes place prior to senility, *i.e*, *pre*-senile osteoporosis. Further, it is questionable whether senile osteoporosis can legitimately be termed an involutional phenomenon. For, the body evolves in a descending line from youth *to* senility, a process which is called involution, by definition, involution ends when senility is attained. This period is difficult to define; it depends on the constitution and cannot be bound rigorously to the attainment of a specific age. The changes that take place *during* senility could, with all due respect to old age, be called decay, disintegration or even incipient mortification. The true meaning of "revolution" (even in the descending sense of involution) does not encompass what is meant by "disintegrating process".

Moreover, to our mind the "involutional osteoporosis" of the literature has a far wider connotation than Jesserer's "pre-senile involutional osteoporosis".

The impression received from the published work of Jesserer and co-workers is that (pre-senile involutional) osteoporosis is admitted only upon clear radiological evidence, or when previously vague complaints suddenly become acutely severe through vertebral infractions Admittedly, "purely subjective"

prodromes are described suggesting rheumatic disorders, which have to be differentiated from the complaints associated with spondylosis deformans (pain on beginning to move after rest); further, tenderness on pressure of the superficial parts of the skeleton, increased tonus of the loin muscles and sciatic pain.

These extreme forms, entailing impression fractures of vertebrae and shortening of body length as well as radiologically demonstrable osteoporosis, afflict only a small proportion of the persons who *suffer* from involutional osteoporosis. This we believe because the process does not become radiographically visible until one-third of the calcium has been withdrawn from the bones (Martin). As calculation of the daily loss of calcium in relation to the total reserves of calcium has shown, under unfavourable circumstances it takes the body at least one year to do this (Martin). It is not when it becomes clearly visible in radiographs, any more than at the moment when other signs of involution become apparent (at about the 50th year) that involutional osteoporosis *begins* to manifest itself as a disease. Jesserer himself points out that the endocrine system in women functions disharmoniously for an appreciable time before menstruation becomes irregular. In fact, this osteoporosis is only one symptom of a transformation that is taking place in several spheres. The calcium impoverishment of the bones is microscopically and biochemically demonstrable prior to the stage at which the disease is discernible in radiographs and even then produces various vaguely defined complaints.

These views are more in line with those held by the first authors writing on involutional osteoporosis, *viz.*, Albright (endocrine disorganisation, ovarian atrophy), Kienböck (osteoporosis of the menopause), Polgar, Schmorl, all of whom summed up the clinical picture itself in less concrete terms than Jesserer's.

This approach has the advantage of being more comprehensive, in that it also embraces subclinical cases. We are well aware that it complicates the already difficult differential diagnosis of vague low back pain by the addition of a clinical picture which defies confident diagnosis *in vivo* for lack of radiological evidence of changes and on account of the practical impossibility of resorting to microscopical examination.

Believing that this malady is very common and recognising the urgency of discovering a timely, effective therapy for the relief of sufferers from it, we here append the plan of treatment devised by Jesserer and Kirchmayer. They deplore the practice of prescribing a large orthopaedic corset and management with a plaster corset or plaster bed, as inactivity exacerbates osteoporosis. They recommend exercises, even if deformities already exist, possibly with

the patient wearing a light *canvas body-belt* which, while offering support, inhibits extreme movements.

Pain can be alleviated by *massage*, *irgapyrine* or local *Novocaine infiltration*.

Hormone therapy is reputed to be very successful, both in clearing up the complaints and as a preventive against malformations. (Testosterone propionate, 25 mg 3 times a week, from the second to fourth week 25 mg twice and finally 25 mg once a week administered intramuscularly.) Testosterone and other similar preparations being suspected of favouring proliferation of predisposed tissue, 10 mg of oestradiol are administered per week, this small dose being deemed sufficient to counteract the theoretically possible carcinogenic action of testosterone. The authors say that, apart from the fact that scientific investigation of the matter (Cooke, Lesser, Tausk and co-workers) failed to provide evidence of this carcinogenic effect, they maintain that, despite this theoretical risk, there is no alternative but to administer testosterone, on account of the threat of invalidism as the result of involutional osteoporosis.

Finally, patients are post-treated for one year with testosterone (50-100 mg) – oestradiol (10 mg) *deposit* once per 4 weeks. The authors consider the administering of calcium or Vitamin D as being useless and even dangerous (Jesserer 1956, p. 60)

Instead of testosterone, we prescribe injections of 25 mg of durabolin (norandrostenolone phenylpropionate) three times a week for one year in diminishing doses.

It goes without saying that everything stated above with reference to an overt, clinically apparent pathological condition likewise applies, in a relative sense, to less patent, so-called "subclinical" cases There is no doubt that these fluid transitions from "feeling well" to "not feeling so well" to "feeling rather indisposed" (Rumke) do exist, how often, can only be guessed at by those who frequently encounter these doubtful cases

We admit the one-sidedness of this review. In relation to a disordered metabolism of the skeleton we have concentrated almost entirely upon a disturbance in calcium metabolism. We have not discussed other constituents of the bone tissue which may well be just as important as calcium (proteins, phosphates and so forth). Nor have we considered here certain conditions about whose influence on calcium metabolism and their practical significance doubt still exists.

We shall merely mention acidosis and alkalosis as factors in calcium metabolism, hyperventilation and abuse of beverages containing carbon dioxide. As the latter, in extreme cases, may lead to a steep drop in the concentration of Ca ions in the blood, to the extent even of producing tetany, chronic excessive consumption of these beverages may not improbably affect the skeleton.

Our argument in the foregoing has been that probably countless middle-aged and older people suffer from low back pain due to as yet undefinable causes.

The known ones, such as hernia nuclei pulposi, anomalies, luxations, inflammations and rheumatoid disorders, could not be detected in these persons, or else could be ruled out with a high degree of probability. Before suspecting these patients of suffering from psychogenic disorders, it is right to consider whether their complaints may not rest upon an organic foundation, which is often impossible and usually difficult to prove.

A word or two about *rickets*. In its common form as a familiar clinical picture it is not dealt with separately. We should, however, recall that, through calcium impoverishment of the bone at a youthful age, rickets is capable of causing many deformities of the skeleton, including a secondary scoliosis.

Although rickets is a deficiency disease and starts when the body is deprived of the necessary quantities of Vitamin D, the need for a minimum per kilogram body weight proves to be endogenously determined. Von Verschuer states that the predisposition to rickets in this sense is hereditary. This put into other words means that there must be some individuals who, by nature, can do with such minimal amounts of Vitamin D as are present in all natural foodstuffs, even the poorest.

The deformities produced by mild forms of rickets are not so serious as to be held responsible for pathological motoricity in the literature. The severe forms are becoming rare. Deformities, if resulting from them, may bring about a pathological posture and pathologically executed movements. Pain in the back and limbs is repeatedly mentioned among the residual conditions of rickets in the literature. Satisfactory explanations accounting for a mechanism through which this pain is produced, are not given.

Anomalies of the Vertebral Arch

The vertebral arch performs several functions, primarily a *protective* one; posteriorly the vertebral canal is enclosed as in a flexible armour. The spinal cord and cauda equina are protected in this way (Lewin, p. 41).

Then, the arch *bridges* the articular processes to the right and left of the median line. Theoretically, this would imply either of two alternatives in the absence of an arch, *viz.*, (1) the vertebral joints would no longer synchronise properly in pairs, causing pain or discomfort, (2) possibly, however, the joints on either side were not built up symmetrically, or were not in line with each other, but were nevertheless forced by an arch to function synchronously in pairs. This distorted movement might cause pain through strain of a joint and in that event the removal of the transverse connection – the bridge – (not an

uncommon operation, because the vertebral joints are quite often congenitally asymmetrical) may well clear up lumbar pain. As low back pain was sometimes





found to disappear after so-called negative explorations (removal of arch), this hypothesis finds some support in practical experience.

No direct *static* function is to be expected of the arches, themselves, as they lie, unattached, one above the other with intervening spaces. But, together with the stretched ligaments in between, the arches are a unified whole; in forward flexion the body is suspended from it, as it were. Thus combined, the arches and ligaments may have a static function. Their position behind the joints of the vertebral column is the main factor preventing a forwards sagging of the body. Any lesion of the connecting ligaments (trauma, wear and tear, laminectomy) of necessity transfers this function to the muscles and fatigue, insufficiency, secondary changes or pain may ensue.

Finally, the arches serve as the site of *attachment* for smaller back muscles.

The vertebral arch is assumed to develop through the fusion of several (symmetrical) bone nuclei, both with each other and with the bone nuclei of the vertebral body. If some bone nuclei are not connected in the normal way, congenital disorganisation in the build-up of the arch will occur. On the other hand, Junghanns and Zukschwerdt (p. 82) accept it as proved that a normally developing arch arises from a single cartilage; hence spondylolysis or spondylolisthesis would result, not from the failure of two bone nuclei to fuse, but from a congenital disturbance owing to which the cartilage of the arch is laid down in two non-fusing parts.

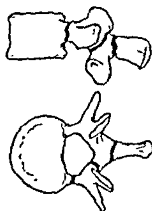
What has been said with reference to the endogenous changes in the vertebral body applies equally to the endogenous character of these disturbances. These are manifested principally by entire or partial detachment of the arch, either on one side or on both, and the complete absence of it.

Changes in the shape of the arches will be discussed later, when we come to deal with the spinal canal

Spondylolysis and spondylolisthesis

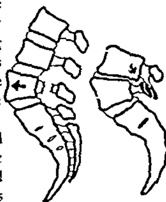
These two disorders receive ample mention in the literature and it is generally accepted that they stand in causal relation to back trouble or radicular pain.

Among others we may mention Armstrong (p. 108), Brocher (p. 39), Epstein (pp. 41, 239, 246), Spiegel (1956, p. 195), Thurel (p. 29). (See p. 436.)



The bony connection in the interarticular process or in the attachment of the arch to the vertebral joint may be incomplete, the effect in a radiograph being as though there were a fissure or pseudo-joint. This fissure is filled with hyaline tissue. It is difficult to establish a traumatic aetiology if radiographs taken just before and immediately after the trauma are not available for comparison. It is assumed that an endogenously caused defect in the interossification of the nuclei of the arch can become manifest through an injury. As a rule, spondylolysis is detected at adult age during a routine radiological screening in an endeavour to account for low back pain. At a youthful age, and certainly at a pre-natal stage, the defect cannot be diagnosed, because the bone nuclei have not yet fused. The fissure in the arch, either on one or both sides, can be differentiated microscopically from a true joint and from a pseudo-joint, e.g., a joint anomaly. There may be evidence suggesting a traumatic aetiology, such as remains of a fracture.

Spondylolisthesis means literally: slipping vertebra (λισθαίνειν, λισθην: slip). Medical parlance distinguishes between vertebral slipping (*e.g.*, laterally as in scoliosis) and spondylolisthesis, *i.e.*, forward or backward displacement. Hyperlordosis is one of the signs associated with spondylolisthesis: when one vertebra is displaced forwards, an adjacent one makes the impression of being displaced backwards. In 82.1% of cases the condition is in the lumbo-sacral region; in 11.3% at L.IV–L.V; in 0.5% at L.III–L.IV and in 0.3% at L.II–L.III (Meyerding, see Epstein, p. 345).



With some exceptions, where the effects of violence are involved, spondylolisthesis is almost invariably the result of strain in the presence of an existing spondylolysis, the fissure widens, the arch remains fixed (temporarily) by the articular processes on the joints, the intervertebral disc drops and degenerates: the vertebral body is displaced forwards.

The word spondylolisthesis is so closely associated with the idea of displacement as the result of a gap or fissure, that the backward displacement of a vertebral body, arch and articular process, as an aggregate, is

called a pseudo-spondylolisthesis (Junghanns, see Schmorl, p. 247). This takes place with a dislocation or subluxation of the vertebral joints and may be the result of any disc narrowing (Brocher, p. 28, Harrenstein, p. 1400, Mulder, p. 1626). Armstrong (p. 102) and Lewin (p. 723) associate this retrolisthesis with the fact that the articular facets of the vertebral joints around the narrowed disc are oblique in relation to the vertical plane. For further details on pseudo-displacements ("pseudo-pseudo spondylolisthesis") see p. 438.

With spondylolisthesis, the angle between the sacrum and the vertebral column is smaller than normal, but, on an average, not smaller than the angle usually found in old people. This is thought to imply, for one thing, that a small angle is not a predisposing factor for spondylolisthesis (Schmorl, p. 239).

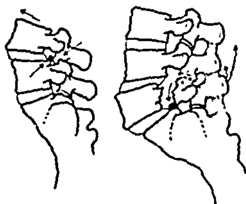
The failure of the nuclei in the pedicle of an arch to ossify is not considered to be only endogenously determined. A defect may also be brought about exogenously by foci of infection and tumours as well as by injury. Willis's opinion that a fissure formed in spondylolysis is always a result of non-ossification of the nuclei, however, is hotly disputed by some who lay more stress on excessive strain on the very young individual and even suggest that the fissure has its origin in natal traumata. The impossibility of bringing such traumatic fractures about experimentally under very heavy strain would seem to argue against this suggestion. Epstein (p. 346) enters fully into the arguments for and against these views. We add to them the statement made by Schinz (p. 1443) to the effect that fissure formation, as implied in this context, has been found in the embryo!

True spondylolisthesis has been observed in a foetus of 40 cm, a baby of 17 months and a child of four years. Bailey, Brocher, George, reported a combination of spondylolisthesis with anomalies in vertebrae and vertebral joints (Epstein, p. 347). The authors believe that heredity is largely responsible and were able to prove that it was in several cases. Bailey noted spondylolisthesis in an average of 5% of cases; in 4.4% he saw a (fissure-like) defect in the arch Only 0.5% complained of low back pain. Brocher (1957, p. 39), on the other hand, estimates that spondylolisthesis is associated with clinical symptoms in 25% to 50% of cases, but he does not believe that the intensity of the complaints corresponds to the degree of displacement.

It is not stated anywhere in the literature that the fissure in the isthmus of the arch is painful. The mechanism causing pain, which comes into operation in the presence of a comparatively large cleft, is dealt with fully from several points of view

The fact that traction of the ligaments occurs due to the shift sufficiently explains why *pain is felt in the back*. Parts of the vertebrae come into contact,

owing to which the periosteum is either stretched or compressed. By local action the muscles will try to prevent dislocation and become exhausted. Where there is considerable dislocation, some changes also take place in the joints and the arch of the vertebra involved: the joints are overburdened in an abnormal position and the arch rotates round the changed vertebral joint. The connection between the arch and the vertebral body having been destroyed, movements around the vertebral joint are no longer inhibited by the body; owing to traction of the muscles at the arch and spinous process, movements of greater than physiological amplitude may take place. Further, the posterior part of the subjacent vertebral body will shift dorsally in relation to the plane of the joint and thus contribute to deformities of the joint.



The most familiar explanation suggested for *radicular pain* is that narrowing of the disc, combined with a backward shift of the posterior rim of the subjacent vertebral body, lessens the diameter of the intervertebral foramen. Moreover, the root, as it runs there, is displaced by this posterior rim and comes to pass over a ridge of bone. If the foramen has become reduced in size, the root will be pent in. Woolsey inclines more to the view that radicular pain is caused rather by rotation of arch, joint and spinous process as a unit. This involves an entirely different mechanism. An argument in favour of his view is that this mechanism already operates in the case of spondylolysis, without vertebral displacement in any serious degree. Emphasis is laid upon the tenuous support given by the fibrous connection in the isthmus of the arch. By whatever forces – these are not specified – the spinous process plus arch rotates about the pivot of the vertebral joint in a cranial direction; the soft fibrous tissue is not capable of controlling this movement. The bony remains of the arch on the anterior side of the vertebral joint also rotate, *viz.*, in a caudal direction, hence in the direction of the root running below it.

In the classical view, the intervertebral foramen is filled up, or narrowed, by the posterior margin of the subjacent vertebra plus disc under a force exerted from a ventral direction, but, according to Woolsey, the compressing mass comes from a cranio-dorsal direction. This view receives support from the fact that radicular pain also occurs in association with spondylolysis and from the relief obtained by removal of the spinous process, arch and joint. (We had similar success by this procedure, without an accessory fusion, *viz.*, in

two cases in which we left a retropulsed disc *in situ* in order to prevent worse to come.)

Opinions differ as to the frequency with which spondylolisthesis occurs combined with hernia nuclei pulposi. Woolsey *never* came across the combination. He cites Briggs and co-workers, who reported one hernia in 25 cases of spondylolisthesis. Other orthopaedic surgeons have told us that they are of the same opinion. Thurel (p. 29) does not refer to the incidence of both abnormalities simultaneously. Armstrong (p. 110), on the other hand, contends that abortive results of therapeutic treatment of spondylolisthesis are often due to failure to recognise a hernia, possibly at a level other than that of the spondylolisthesis. Schmorl thinks that it is precisely through changes in the disc that a vertebral body shifts and adduces as evidence radiologically observed narrowing of the intervertebral disc involved in the majority of cases of spondylolisthesis. Meyerding reports hernias in 6% of cases. Hagelstam (p. 141): "Pain syndromes and neurological findings . . . are not due to the retroposition as such but, obviously, to degeneration with or without herniation of the disc".

The symptoms produced by spondylolisthesis can be summarised as follows: low back pain, 61.3%; low back pain plus radicular pain 17%; paralysis, 1.9% (Meyerding). The neurological syndrome, as well as the complaints, may be identical with those of hernia nuclei pulposi.

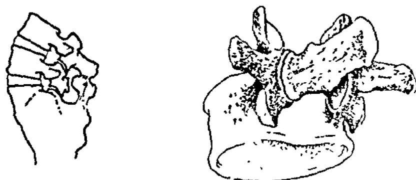
According to Thurel (p. 29), chronic bilateral complaints point very strongly to spondylolisthesis, but Armstrong (p. 108) states that the symptoms may be either unilateral or bilateral. He attributes these to the occurrence of some rotation, owing to which the root on one side may be more affected than that on the other. His basic assumption is obviously that the defect exists in one half of the arch only, through which rotation becomes possible. Reference is made in the literature to bilateral defects of the arch isthmus, but generally no mention is made as to whether they are unilateral or bilateral. We sought in vain for a clear pronouncement on the matter.

Is it right to ascribe the one-sidedness of radicular symptoms associated with spondylolisthesis to rotation of the lumbar column? We are inclined to believe that this conclusion is premature, not to say altogether incorrect. Without appealing to our own material, we may point out that no rotation is apparent in the radiographs reproduced in every manual or monograph; *e.g.*, in Armstrong's own book. Add to this that considerable variety occurs in the structure of the spinal column within normal anatomical limits, for one thing in symmetry. This is more conspicuous still in an abnormal spinal column, of which spondylolisthesis is, after all, one aspect We know, moreover, from the literature that spondylolisthesis is often associated with other abnormalities

or anomalies. Accordingly, we believe that *congenital predisposition* of one of the two lateral halves of the vertebral column offers an acceptable explanation for unilateral radicular symptoms of spondylolisthesis which, on radiological evidence, is not associated with rotation of a lumbar column. Asymmetry is a common congenital "anomaly", often occurring in combination with other congenital changes in shape. Owing to this asymmetry, the room to spare on one side of the spinal canal – say at the site of the root in its extrathecal course – may be smaller than on the opposite side; naturally, therefore, symptoms of root compression are more likely to occur there.

The unattached arch

If we take the lumbar vertebra to be a single functioning unit, we can quite simply compare it with a lever. We have the vertebral body lying ventrally, the arch and the spinous process dorsally and, in between them, the fulcrum of the lever, *viz.*, the joints. With dysjunction in the pedicles between the vertebral body and the joints (spondylolisthesis) the body begins to slip off and the arch plus spinous process may carry out rotary movements. If there is dysjunction in the posterior part of the arch between the spinous process and the joint, the body remains in its place while the arch plus spinous process are unattached. Clearly, the consequences of the latter are far less dramatic; it might be inferred from the scanty reference to it in the literature that the unattached arch is overlooked.



In 1953 Hanraets reported the syndrome of the unattached arch L.V as the result of congenital true joints in the arch itself.

These were situated on either side between the attachment of the spinous process and the articular process or the vertebral joints. The pathologist Weyers recorded true, normal joint facets. The patient complained of low, indeterminate pain upon dorsiflexion, *i.e.*, he was able to bend forward without pain, but every backward move-

ment of the back, *i.e.*, from forward flexion, stooping and the erect posture, especially hyperextension, caused local pain. There were no radicular symptoms. Upon palpation, the L.V spinous process was felt to be slightly prominent. The pre-operative radiographs revealed fissuring only in the left half of the arch, for which reason the case was provisionally diagnosed as primary or post-traumatic lysis of the arch. This observation was followed by others.

In his monograph, "The Back and its Disk Syndromes" (1955), Lewin describes (p. 488) the occurrence of the unattached arch as the result of anatomical variations, *viz.* defects in the joints, incomplete asymmetrical arches and defects. The arch may be entirely or partially unattached: through a new joint of its own (Hanraets), a joint anomaly (Lewin-Willis), endogenously through lysis (Lewin-Willis) or exogenously after an injury (Mensor). Surveying this pre-operative, often misjudged phenomenon as a whole, we find that its occurrence is not rare; it is come upon incidentally during laminectomies.

Spina bifida occulta

Apart from morphological abnormalities and defects, the most manifest malformation of an arch is its absence. The adjective "occulta" refers to its exterior invisibility, *i.e.*, the 'hidden" malformation. The term spina bifida is also used to denote a condition in which the arch remains open, its associated external manifestations, however, being visible. This implies a misunderstanding, *viz.*, that the arch is split; but the split itself is not visible on the outside. The term is misused, in the sense of a part for the whole, as applied to a condition which, to avoid being misleading, could be better described as "meningo(myelo)cele".

The confusion to which this terminology may give rise is illustrated by the fact that in one manual Moor's statistics of 1905 are quoted in reference to spina bifida occulta, while in another they are cited in connection with meningocele! Departing from usage, we shall adopt the nomenclature spina bifida = open arch, here and in the ensuing chapters

An arch may either be lacking altogether, or be split; a partial defect is usually situated in the median line (66%) or laterally (33%). The defect may be clearly manifest or only indicated, in the latter case it cannot always be established with certainty radiographically, but can, on the other hand, be verified at operation. Epstein (p. 59) states that the radiographic detection of a spina bifida depends on the angle of the pelvis at which the radiograph is made, a tilt of 45° will reveal abnormalities which did not at first appear on the radiographs.

In view of the variety and multiplicity of the intermediate forms, statistics on the frequency of the condition cannot be expected to agree very closely.

Southworth and Bersack		18%
Dittrich		5%
Buck Hillsman, Bason		6%
Friedman, Fischer, van Demark		36%
Epstein (see survey p. 99)	roughly	36%
Lewin (p. 54)		17%
Schinz (p. 1429)		20%
Willis		6—10%
Material Ursula Clinic	roughly	19%
(in conjunction with a slipped disc		32%)
(in conjunction with a wide lumbar-sacral canal		23%)

As Curtius and Lorentz (1934) say, the incidence of spina bifida in the literature varies between 3.6 and 48%! The conjunction of enuresis with spina bifida was 59.5%.

According to Epstein (p. 98), spina bifida occurs more often in the fifth lumbar vertebra than in S.I. As against this, we have

Southworth and Bersack	S.I	16%	L.V	2.2%
Lewin	S.I	11%	L.V	6 %

Spina bifida also occurs in the cervical, thoracic and high lumbar parts of the spinal column, but its frequency there is negligible in comparison with the percentages given above.

There are numerous combinations, especially with the low sacral spina bifida (open sacral hiatus). The radiographic aspect of spina bifida is known: no arch, a fissured arch, the crooked or maybe asymmetrical malformation of the remains of the arch. These defects, if of a minor or very minor nature, might be overlooked, especially if the examiner minimizes the significance of spina bifida as a factor in relation to the complaints for which the radiographical examination was carried out.

The consensus of opinion in the literature is that spina bifida is often associated with numerous anomalies elsewhere in the body, of which we shall here specially mention the radiographically visible transitional vertebra (Brouwer, p. 441), cervical ribs, basilar impression and atlas assimilation (De Vet), rachischisis "totalis" (De Jong), malformations of the vertebral bodies and spinous processes and, in particular, malformations of the vertebral *joints*, also many other stigmata degenerationis (Sillevis Smitt). Walker, also quoted by Lewin (p. 54), was the first to report dilatation of the spinal canal in association with spina bifida, but it is not clear whether he meant that lipoma was always found simultaneously in the spinal canal.

Of the defects not usually directly apparent in radiographs, which often



Spina bifida occulta of L V Rachischisis sacralis totalis. Flat oval wide spinal canal. Spondylolysis, spondylolisthesis

occur at the same time as spina bifida, we mention anencephaly, hydrocephalus; Arnold-Chiari's malformation (Van Houweningen Graftdijk); hydromyelia, amyelia, diplomyelia (Folkerts), diastematomyelia, myelodysplasia of Fuchs, meningo-myelocele (anterior), cysts (in the spinal canal, Brouwer, p. 445).

Finally, tumours, such as fibroma, lipoma, epithelial cysts and teratoma, are quite often encountered in association with spina bifida (Brouwer, p. 435, Folkerts)

The distension of the spinal canal referred to above will be found to be exceedingly significant when we come to define the degenerative or weak back in the following chapters. We shall therefore revert to it *in extenso*, as also to Sarpyener's statement that spina bifida may be associated with a lamella-like narrowing of the vertebral canal. Varieties in this sphere can only feasibly be dealt with in an overall survey.

The literature reveals anything but concurring views on the *aetiology* of spina bifida. The obvious association of this defect with other congenital anomalies,

the endogenous character of which is generally accepted, provides strong support for the geneticists' contention that spina bifida is endogenous in origin. The same interplay between the genes controlling morphology is said to be operative here as that which produces the malformations of the vertebral body. (Bauer and Bode. Ingraham and co-workers were able to show that spina bifida was hereditary in 16% of 277 cases, while other anomalies as well occurred in the families of 15% of these persons.)

Tracing in broad lines the general opinion that spina bifida is endogenous, we find that nevertheless very contrary views are held as to the *mechanism* by which this malformation is accomplished. In 1940, Brouwer could not agree with the then current view that spina bifida is due to a local failure of the medullary plate to close completely into a neural tube. The failure was ascribed to exogenous (toxic, chemical, mechanical) causes, some researchers assuming that there was also an endogenous cause, *e.g.*, that the cells of the medullary plate possessed inadequate formative capacity. Excessive secretion of cerebrospinal fluid, causing, it was thought, hydromyelia, was considered to be highly significant. The theory that inflammation had to be reckoned with as an important factor was countered by the argument that it does not occur, or, if so, very rarely. Thus, hydromyelia is also merely an incidental complication.

Brouwer (p. 436) sums up as follows:

"Reviewing, in the aggregate, the available data from which a theory as to the pathogenesis of spina bifida could be formed, one is forced to conclude that no single cause, by itself, is wholly responsible for this defect. It is only when it is viewed superficially that the picture of spina bifida creates the impression of being a systemic disturbance. It is variation which at once strikes the observer who examines his material closely under the microscope; it seems clear that endogenous as well as exogenous factors must be at work. Again and again, during my study of this malformation, it has struck me that, among other things, regressive changes due to purely pathological foetal processes must be involved. The assumption that the neural tube is imperfectly closed does not lead us very far."

Keen is another who has many objections to raise to the "open neural tube" theory. He champions the view, in support of which he advances many arguments, that all congenital deformities (including anencephaly) complicating spina bifida must be traced to disturbances in the secretion of cerebrospinal fluid and raised fluid pressure. If hydrocephalus *internus* develops from this raised pressure, the nerve tissue will be destroyed (anencephaly, hydromyelia) and the dorsal plates proximate to the neural tube will cleave asunder or lean apart (spina bifida and myelocele). If the raised pressure produces only

hydrocephalus *externus*, the dorsal plates will only distend, partly, entirely or temporarily (spina bifida, meningocele). *(Wide spinal canal; the present author's addition.)*

Hence the splitting of the neural tube is secondary in these cases; simple spina bifida and meningocele need not involve gross malformations, which, indeed, is borne out by experience. Minor changes may, however, be a secondary result, or are possibly induced by the same (endogenous) factors as those responsible for the disturbance in the circulation of the cerebro-spinal fluid. Finally, these minor disturbances of the central nervous system (De Lange, see Brouwer, p. 435) may be the primary cause of the disorganised fluid secretion, the mechanism of the raised pressure, entailing cleavage of the neural tube and membranes, etc., coming into operation subsequently. It is pertinent, too, at this juncture to quote Brouwer once again: "We have learned from microscopical examination of the central nervous system in cases of spina bifida that not uncommonly changes have also taken place in the brain . . . Professor C. de Lange's examination of the central nervous system led to the discovery of other developmental disturbances in cerebro *which had culminated in hydrocephalus.*"

The theory developed by Keen, Professor of Anatomy at Durban (South Africa), possesses the attractive quality of being generally explanatory and of resting on sound arguments From the notes with which he most generously provided us, we feel that we should set down the following arguments *in extenso*.

1. The theory postulating that "the neural tube *remains open*" of necessity implies that all similar congenital defects must have arisen before the third to fourth week, as the embryonic neural tube is already closed after this period; hence the mechanism keeping it open must become operative prior to the fourth week. Keen points out that the failure of the neural laminae to fuse would constitute such a fundamental disruption to an embryo 3 mm in length that it would cease to develop any further. The normal development of the peripheral nerves (in the cases described) makes it look as though the nerve cells of the spinal cord or of the ganglia continued to function up to a comparatively late period of intra-uterine life (These observations are consistent with those of Bauer and Bode and of Sokolansky, who likewise found the muscular and peripheral nervous systems to be normally developed in cases of anencephaly and amyelia.)

2. Myelocele, anencephaly, etc are always associated with a tendency to nervous degeneration. The "patency of the neural tube" does not satisfactorily explain the cause of this degeneration By contrast, it is easy to

imagine the effect of raised fluid pressure upon a closed neural tube and on the closed system of membranous sheaths around the central nervous system.
3. The fissuring and protrusion tendency associated with some defects of the vertebral column or skull applies only to the cerebral and spinal *membranes*, whereas the organs themselves appear to be normally formed. The adherent of the "patent neural tube" theory must necessarily assume that in those cases the walls of the neural tube are closed secondarily later on.
4. Gradations between a closed neural tube and meningocele or spina bifida have not been encountered in experimental embryology. Keen accounts for reference to them by the confusion in manuals which has resulted from the incorrect use of the term spina bifida. It is sometimes used, erroneously, to imply non-closing of the neural laminae, whereas the essential meaning of the term is "open arch" or, metaphorically, meningo-myelocele and meningocele. (Keen has ascertained by experiment that no fully developed foetus ever comes into the world with the neural tube still open.)
5. Rings of tissue around the primitive neural lamina, which are often observed in association with anencephaly, are almost certainly remains of the dorsal cerebral membranes which have burst open. The presence of these tissue remains can only be accounted for on the basis of the raised fluid pressure theory.
6. Previous authors divided the deformities complicating spina bifida into two groups, one group being said to be the result of failure of the neural plates to fuse, the other being the result of raised fluid pressure. Keen prefers to postulate one common genesis for all these essentially similar congenital deformities, rather than to assign a different cause to each, requiring two different pathological mechanisms.

Keen might, moreover, have referred to the incidence of pre-natal hydrocephalus internus communicans, for this proves that disorganised production of fluid can exist in the embryonic stage.

It is clear to us that he does not lose sight of the possibility of an endogenous factor underlying this mechanism. As an anatomist, he does not express an explicit opinion on the matter and leaves the possibility of an exogenous cause affecting the embryo an open question.

After an extensive study of the literature and impressed by the conviction with which a researcher like Brouwer declares that he was unable to detect any signs of an exogenous cause, we are all the more inclined to accept the results of the geneticists' investigations.

Precisely because, in a few very incidental cases, he, hesitantly, recognises a possible connection between this defect and a mother's attack of influenza,

syphilis or chronic abuse of alcohol, the question arises as to whether this defect as a whole can legitimately be regarded as a *degenerative* feature. For it appears that a given pathological condition can – be it sporadically – induce the same signs and symptoms which occur endogenously in a certain group of the population; this occurs with manifestations which change the body in a retrogressive sense and this offers an appealing argument for likwise stigmatising manifestations like spina bifida as degenerative.

We attach much importance to Keen's theory. Presumably this anatomist had no opportunity of trying out his ideas in the clinic. Since the surgical treatment for hydrocephalus in very small children by drainage systems has been resumed, it has transpired that the production of cerebro-spinal fluid and the raised fluid pressure associated with it fluctuate considerably. In fact, the drainage treatment is applied in the hope that the raised fluid pressure will only occur temporarily in the treated case and that later the child will no longer need the fluid-carrying drain. In roughly 25% to 50% of the cases that hope is fulfilled. If this observation is referred to the embryonic period of life, it would not be inconsistent to suppose that the production of fluid fluctuates at that time as well; the fluctuations might be temporary, say until the blood-serum barrier has adjusted itself. If there is no adjustment, the excess production becomes permanent and pathological conditions, or deformities, set in. If the over-production is only temporary or of minor degree, the changes brought about are likewise of a minor nature. The latter may well be "subclinical", *i.e.*, changes that are not associated with clinically detectable or easily recognised signs and symptoms; they will probably be noticed by chance, or incidentally upon examination of the patient for quite different complaints. One of these subclinical forms might be spina bifida occulta or the wide lumbo-sacral spinal canal.

We have one more point to raise within this context which still holds so many open questions. Why (according to Keen) should hydrocephalus externus develop on one occasion and hydrocephalus internus on another? Why at one time a spina bifida at S I, whereas at another it is at L.V?

If we can be confident that there is no evidence of exogenous causes of these deformities, then, to resolve the above problem, we must assume that there is an endogenously determined centre of least resistance.

Kuhne (Erbbiologie III, p 199) and Schroder sum up this view in the following sense · Hereditary deformities are not *locally* heritable. Abnormalities of a developmental factor of the whole vertebral column reveal themselves in a given individual in the form of spina bifida. Disorganisation of the basal developmental factor also interferes with the work of the independent

gene which controls the fusing of the mesenchymal and ectodermal plates.

Seen in this light, under certain circumstances spina bifida might be induced by a previous malformation of the nerve tissue; *e.g.*, Fuchs' myelodysplasia.

Keen's theory undoubtedly offers an explicable mechanism clarifying the anatomical origins of these deformities; but both the deeper aetiological factors, which also underly this mechanism, and the predilection for place and form are still outstanding problems for which there is no answer known at present other than the vague idea of "endogenously caused".

Meningo-myelocele, hydromyelia and diastematomyelia are deformities of the spinal cord liable to accompany spina bifida (occulta) (simplex); text-books describe them in detail and they are not of special interest to the student of the function of the back.

Other malformations or anomalies of the spinal cord, dura and cauda equina, which often occur in conjunction with spina bifida, are described in another chapter. Fuchs' myelodysplasia is reverted to in the chapter dealing with pathological motoricity.

Outwardly the cleavage of the neural arch is sometimes accentuated by *hypertrichosis* (satyr's tail) at the site of the lowest lumbar vertebrae. This is not to be confused with the abnormal growth of hair sometimes encountered when a patient has been shaved on an earlier occasion for a lumbar puncture. It is the more likely to be misleading because these patients, in particular, on account of the indeterminate character of their complaints, are apt to go from one medical adviser to another and sometimes deliberately omit to mention a previous examination. Naevi, accumulations of pigment, absence of pigment and local cutaneous changes, as in scleroderma, also occur in the skin above the cloven arch. They are to be regarded as a manifestation of trophic disturbances due to root disorders (Brouwer, p. 439.)



Sinus pilonidalis

The presence of spina bifida may be betrayed by retractions of the skin, which should be differentiated from anal fistulas, subcutaneous fistula pathways and a rudimentary communication between the skin and dural sac; the latter is called *sinus pilonidalis* and is treated along with dural deformities. In association

syphilis or chronic abuse of alcohol, the question arises as to whether this defect as a whole can legitimately be regarded as a *degenerative* feature. For it appears that a given pathological condition can – be it sporadically – induce the same signs and symptoms which occur endogenously in a certain group of the population; this occurs with manifestations which change the body in a retrogressive sense and this offers an appealing argument for likewise stigmatising manifestations like spina bifida as degenerative.

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We have one more point to raise within this context which still holds so many open questions. Why (according to Keen) should hydrocephalus externus develop on one occasion and hydrocephalus internus on another? Why at one time a spina bifida at S I, whereas at another it is at L.V?

If we can be confident that there is no evidence of exogenous causes of these deformities, then, to resolve the above problem, we must assume that there is an endogenously determined centre of least resistance.

Kühne (Erbbiologie III, p 199) and Schroder sum up this view in the following sense: Hereditary deformities are not *locally* heritable. Abnormalities *of a developmental factor of the whole vertebral column reveal themselves* in a given individual in the form of spina bifida Disorganisation of the basal developmental factor also interferes with the work of the independent

spinal column or an acute infectious disease may bring such symptoms into the open, there is usually no apparent cause. It is highly probable that adhering membranes are unable to follow the growth of the individual, with strong traction upon the roots and the spinal cord as the result. The *increasing size of tumours* or *cysts* that may be present also possibly has something to do with it." (Brouwer, p. 442).

Clearly, the therapy adopted aimed at cutting through these membranes which, at that time, were the only pathological substrate. It is not our intention to imply that observations such as those published by Romback in 1905, Hoelen in 1923 and De Vries in 1928 are no longer made today. Jones and Love (Spiegel 1957, p. 321) report on six patients with a 'tight filum terminale' in a number of 442 patients with spina bifida occulta. In his monograph of 1955 (p. 649) Lewin still cites some of Kleinberg's cases of 1952 and Garceau's of 1953 (Epstein, p. 117); he there refers to the "filum terminale syndrome" which is said to occur, not only in conjunction with more complicated malformations, but also "associated with *simple* spina bifida occulta"; here we quite easily recognise the syndrome of the "persisting membrana reuniens". Both syndromes are caused by inhibition of the physiological ascensus spinalis, or "the distal migration of the spinal column". The same train of thought accounts for complaints and symptoms, including stretching of the roots, displacement of the lumbar ganglia from their foramina (Zukschwerdt), Arnold-Chiari malformation and hydrocephalus. (Bijl, p. 115.)

Barry and co-workers studied the possible factors in the development of the Arnold-Chiari malformation (1957, p. 285). Traction and stretching *via* roots or spinal cord take place if, owing to a primary defect, the myelum becomes fixed and thus impedes the ascent. They noticed, however, that abnormal growth had taken place in certain parts of the central nervous system (midbrain, rhomb-encephalon, cerebellum, myelum) in association with various malformations. This overgrowth produces a protrusion of the hindbrain into the cervical canal. Depending on circumstances, this results in hydrocephalus or an Arnold-Chiari malformation. Overgrowth limited to the spinal cord might produce a myeloschisis and other malformations.

We cannot help thinking that the mechanism in which traction may be the cause of malformations is credited with far too much importance. Experience gained in thousands of laminectomies has since shown how seldom an unambiguous persisting membrana reuniens occurs. It is precisely the later authors, especially neurosurgeons, who had themselves not observed anything unusual in the filum terminale, nor observed a membrana reuniens at countless laminectomies, who are inclined to believe that sufferers from back complaints

with such retractions – fovea coccygea – one may find a small angioma, telangiectasis, lipoma, fibroma and even teratoma.

There is no agreement of opinion at all in the literature as to whether spina bifida involves back complaints and, if so, how these arise.

Let us complete the survey as made by Brouwer (p. 436) and then briefly refer to some other authors

Neurological symptoms may be associated with *simple* spina bifida (occulta), they result from primary changes in the spinal medulla and the roots, they are partly caused by the pulling and pressing of adherent ligaments. The most important signs and symptoms he mentions are club feet, paralysis of the abdominal muscles and of the legs, oedema, trophic disturbances, shortening of the leg, the complete picture of Fuchs' myelodysplasia, retentio urinae et alvi, as well as incontinence and nocturnal enuresis. Let it be well understood that, as he himself expressly states, he observed all these grave disorders in association with the cloven arch, without meningocele or myelocele It seems to us that, later on, he did not persist in this differentiation. Again at a later stage he emphasizes the occurrence of painful sensory disturbances, particularly in the region of the sacral roots, in association with simple spina bifida, when pain is experienced in the lowest segment of the vertebral column with referred radicular pain. (In view of the period of these publications (1940), one wonders whether Brouwer overlooked the then less familiar hernia nuclei pulposi in some of these patients; also those congenital anomalies which "cause" pain, to be dealt with later.)



Membrana reuniens with a very thick S 1 nerve root

It is a striking fact that it is precisely the authors of the pre-hernia period who clearly associate spina bifida with complaints of pain; they usually account for these and other neurological symptoms by increasing traction of the membrana reuniens, the filum terminale and other ligaments[1] (Leri and Delbet; Cossa, p. 218.) It is said to be "... a striking fact that the neurological symptoms need not necessarily be present at birth, but become apparent at a later age, usually in adolescence. Although injury to the

spina bifida... c'est certainement une rarité. Sa cause doit être cherchée ailleurs: *déséquilibre lombo-sacré*, scoliose, arthrose, hernie discale etc."

With this the problem is reduced to that of a disorganised function of the skeleton, *viz.*, pathological motoricity and pathological posture.

In this matter we subscribe to Lewin's point of view (p. 54): "Congenital anomalies are important in laying the groundwork for subsequent mechanical trouble."

Changes in the Joints Associated with Low Back Pain and Radicular Pain

Of all the disorders affecting the spinal column, those involving the joints are most commonly considered to be the cause of back pain. There is no ambiguity about this in the literature. The mechanism causing these complaints is simple and the changes taking place in the joints are *ostensibly* quite clear. Yet it would be difficult to pick out a segment of the spinal column on which opinions are more divided or misunderstandings more rampant than precisely on the joints.

Departing from the plan hitherto followed, we shall first list the possible changes in the vertebral joints, then discuss the lesions known without doubt to be caused exogenously and then, primed with these facts, turn to the literature, with special reference to the endogenously caused disorders.

Arthrosis deformans is said to be the most common cause of invalidism. Schreuder drew up a list of the diseases which reduced the inhabitants of a home for the aged to invalidism.

	Women	*Men*	*Total*	%
Arthrosis deformans	126	42	168	32.81
Hemiplegia	43	28	71	13.86
Heart and vascular diseases	39	18	57	11.13
Kyphosis	7	—	7	1.35
Chronic rheumatoid arthritis	26	10	36	7.03
Diseases of the air passages and lungs	10	9	19	3.9
Other maladies	111	43	154	30.07
	362	150	512	

The first item includes the arthrotic changes to all joints in the body. Comparisons are not infrequently made in the literature between pathological changes in the big joints elsewhere in the body and those in the small joints. The pressure conditions and mechanisms are not the same. The principal

and radicular pain very often have a spina bifida; but they are almost unanimously convinced that it is not the spina bifida itself, but *ancillary* mechanisms which produce the complaints in *these particular* persons. What they recognise, therefore, is a relation in frequency, probably a constitutional relation, but not a causal one.

Armstrong (p. 138): "It seems unlikely that a spina bifida alone is a cause of low back pain and symptoms probably only occur if the bony defect is associated with abnormalities of the meninges, spinal cord or cauda equina".

Epstein (p. 102) is of opinion that spina bifida occulta does not usually produce specific complaints.

The consensus of opinion nowadays is that spina bifida (and congenital anomalies generally) need not be associated with complaints or symptoms. This has been borne out by extensive radiological examinations made upon groups of persons who had never suffered from back trouble at all. It has come to be realised, however, that various individuals who had this congenital malformation begin to develop symptoms in adolescence (Brouwer) or in middle age which could be ascribed to no other cause.

It used to be thought that the pains experienced in adolescence and especially during periods of strong bodily growth were produced by the stretching of ligaments, membranes and so forth; the increasing size of concomitant cysts or benign tumors was also held to be responsible and it constituted a rather forced answer to the problem of the appearance of the symptoms at a maturer age! After all, the membrane theory did not fit very comfortably into the picture of symptoms arising at a period long after the body had ceased to grow. The current tendency, however, is increasingly to stress a consitution predisposed to complaints, of which these malformations are one manifestation. Trauma, inflammation, oedema (Brouwer) and intervertebral disc lesions are mentioned as accessory factors in the syndromes. Latterly, emphasis is being *again* laid on arthrotic or spondylotic changes; apparently minor posterior lipping of the vertebral bodies may reduce the only just physiological dimensions, determined constitutionally by anomalies, to pathological ones. (Briggs, Goslings, Hadley, Hanraets, Lindblom, Magnuson, Williams.) And through these again, whether or not at the time of an injury, strain, inflammation, etc., the complaints are brought on which it was formerly customary to ascribe to the malformation itself.

Others, again, point to the lamella-like widening of the interpedicular distance which is often seen in association with spina bifida and, by virtue of the same mechanism, may take up room (Sarpeyener, Hanraets). Sicard puts the problem in the following words "Le syndrome douloureux attribué au

and tear are to be seen in all vertebral columns; the joint facets are worn down in varying degrees, starting in the 20th year (!) but normally not assuming pathological proportions until the 60th. They speak of degeneration when the vertebral joint of a person of about 30 years of age looks like that of someone of 60. According to these authors, it would appear from the literature that joint cartilage is not, or virtually not replaced after the twentieth year. Put into other words: the individual has to put up with the cartilage which he has at the age of 20 for the rest of his life. From this alone it follows that an advanced process of wear and tear is irreversible. They find certain joint facets worn down in their specimens, but also an over-all thinning of the cartilage; this, indeed, may become so thin that the bare bone underneath shows. Further abrasion of the facets may so weaken the structure in places that the least extra strain or a minor injury will result in lines of fracture. Some of these in their specimens were partly fibrotic. They also found loose fragments of joint. They saw subluxations which had obviously caused a fracture either of part of the joint, or of the cartilage line. Although the cartilage itself does not degenerate, fibrosis of the fracture borders and at the back of the thinner places does take place. Here tissue can also invade the bone. The edges of the worn cartilage begin to curl over and as such may become the precursors of osteophytic growth such as is seen in an advanced stage of spondylosis. Calcification of tissue and osteophytes produce the malformations which show up on radiographs. Finally, Harris and MacNab also noted that the ligaments around the joint change. Cartilage and tissue are deposited in them and the joint could therefore become pseudankylotic. The slightly more distal ligaments might also change correspondingly and signs of degeneration were also recognisable in smaller bundles of muscles. Their material included thickened and swollen joints as though recently inflamed.

The *radiological manifestations* of spondylarthrosis are known and are repeatedly seen in routine examinations, even in patients whose complaints had not suggested the disease (Epstein, Franulovic, Schinz, Van der Meer and many others.)

Whatever the views held as to the *aetiology* of this process, there is little divergence of opinion on its mechanism. Briefly it is: wearing down of the cartilage, whether pathologically changed or not, bone reaction to counteract this process of abrasion, sclerosis at the periphery of the vertebral bodies to immobilise the joint and secondary reactions. In a word, the mechanism by which spondylarthrosis becomes operative is an abrasive process with subsequent reactions. Opinions differ, however, as to what initiates this process. The effect of a direct trauma is known and fractures of the vertebral joint

function of a joint is to permit movement; but, apart from this, a joint may be called upon to give support; furthermore, a rotating, shifting, gliding movement can be made, in which case the function of these joints is rather to conduct movement than anything else. More or less pressure will come to bear on the facets of the joints, depending upon whether the levering action which occurs in a joint is placed more or less favourably as a fulcrum for the forces exerted. According to Milton and Zukschwerdt, per 1 sq. cm facet, the weight borne by a vertebral joint is one hundred times greater than that which a knee joint has to bear. Armstrong calculates that the forces acting upon a joint of the vertebral column are magnified about 15 times by lever mechanism. Hence, it remains to be seen whether it is apt to compare the pathology of these differently functioning joints at all.

We have already had occasion to refer to *spondylosis* while dealing with the vertebral bodies. This is the name given to changes in the vertebral margins caused by the growth of osteophytes (Zukschwerdt, p. 80). The bone becomes sclerotic locally. Hooks, known as parrots' beaks, are formed by new growth. Though there may be some association with the joints, essentially spondylosis has nothing to do with them; it may, however, lead to spondylarthrosis if bony hooks form at the margins of the cartilaginous joint or at the edges of fractures running right across the joint.

There is a known contrast between spondylosis and spondylitis. The latter may be the result of either specific or non-specific infections. An almost identical contradistinction exists between spondylarthrosis and spondylarthritis.

In *spondylarthrosis* changes are found in the bone tissue which are fundamentally the same as those associated with spondylosis. If inflammatory mechanisms are observed in conjunction with them, the pathologist calls the disease spondylarthritis. *The latter* involves inflammation of a true joint, which the pathologist is able to recognise as such. Intermediate forms are significant for the aetiology, as rheumatoid arthritis, for instance, may bring about deformities which could be regarded as secondary arthrosis. Harris and MacNab carefully dissected the vertebral joints of 123 vertebral columns chosen at random at autopsy. Their findings are of the first importance. One of the difficulties involved in this investigation, they say, is the impossibility of forming an authentic opinion on lesions of the vertebral joints, because they are only presented in one single stage per case for examination. The pathological substrate of transitions from one conditon to another at various periods in the life of the same patient is unknown. The authors point out that the cartilage has a certain thickness, varying from individual to individual. Signs of wear

Other constitutionally determined conditions are over-sensitiveness to infection, or sensitiveness to conditions inducing allergy, on the basis of which an allergic arthritis may serve as the springboard for secondary changes.

It is necessary to assume all these constitutional factors, as indeed they are in the literature constantly, to account for those cases in which arthrosis occurs under special circumstances. It is not enough merely to call it trauma or infection, possibly with the added flourish of "rheumatoid arthritis" and "focal infection". Nor is it commendable to divert the train of thought from constitutionally determined special circumstances by stigmatising the quest for an anchorage in the constitution as "taking flight in the constitution".

To what extent are the above views reflected in the literature? Harris and MacNab report the proliferating forms in which spondylarthrosis occurs. A review of the radiological abnormalities is to be found in Epstein and Schinz. Schmorl (p. 213), who again emphasizes the difference between spondylosis and spondylarthrosis deformans, points out that changes do not take place in joints without movement. Spondylarthrosis predominantly attacks the mobile lumbar and cervical vertebral column. Prolonged rest slows down the proliferating changes. When rest is imposed by necessity immediately after an injury, the proliferations do not yet occur, but begin to be active when the patient is again mobile (Schmorl, pp. 209, 242). Lubberhuizen (Sleeswijk, p. 625) states that osteo-arthrosis of the vertebral column was generally regarded as a process of abrasion and was adversely affected by prolonged heavy manual labour. Van Assen found wear of the cartilage facets in all persons above 50 and thought that between the ages of 20 and 40 its occurrence was "only" 60%. Abrasion may be confined to some particular part of the cartilaginous joint; this he attributes to differences in vascularisation. These differences might arise from locally unfavourable static conditions. Schmorl (p. 211) – quoting Heinrich and Stettler – formerly estimated the time that has to elapse before clearly marked changes develop at the margins of vertebrae and joints as 1½ years. But, after injury to a vertebra, he says, incipient marginal proliferations have been observed 4 to 8 weeks later, which must have started within this time. He is very insistent upon this and dwells on the importance of discovering whether a spondylarthrosis is or is not of traumatic origin.

Van der Meer considers arthritis to be the cause of arthrosis. His investigation was intended to show that in the old views, according to which arthrosis deformans was caused by constitution, degeneration and infection, chief emphasis should be laid on infection as the most common cause of these changes in joints. He sees ordinary inflammation, rheumatoid arthritis, allergic conditions which may likewise cause arthritis, as the foundation from which

have been observed radiographically and at operations. This is a question of the patient who was perfectly healthy in youth, whose joints appeared to be normal on radiographs and who, after a serious injury, develops local spondylarthrosis. It is not known how serious the trauma has to be, but it is certain that this process also takes place after chronic strain; a patient who suddenly changes over to different employment may in a comparatively short space of time be suffering from spondylarthrosis in part of the spinal column. A pathological change elsewhere in the body – *e.g.*, ankylosis of a knee joint – may take place in another patient; within a few years the physician may find a localised spondylarthrosis.

It is common knowledge that *inflammation* is capable of producing arthrotic changes secondarily. All the signs of inflammation of a joint may then be found, say, at operation, which the pathologist has no difficulty in establishing beyond doubt, while besides there will be sclerosis of the bone, hooks and so forth, which are to be regarded as secondary.

The literature looks upon rheumatoid arthritis as one of the causes of spondylarthrosis, but the pathological substrate cannot be clearly differentiated. The most cautious view is that rheumatoid arthritis is capable of producing secondary changes.

There is unanimity on the precipitative effect of *strain* on spondylarthrotic changes. It is less a question of any doubt about it than of the significance attributed to this effect. With some exceptions, the idea of undue pressure is always relative, for, when is the strain too great in proportion to the resistance which certain organisms can offer? On the one hand, therefore, the weight of the forces acting upon a given joint will have to be considered and, on the other, the condition of the joint itself. Obviously, a joint that has been changed by an injury is liable to be overburdened locally.

If the changes in a joint are due neither to trauma nor an infection, but to a congenital defect of the joint surfaces, then even a normal movement might put that joint under excessive strain. In the same way, normal movements and a normal static relation could induce pathological changes in the cartilage if this were congenitally inferior.

Finally, if the joints are of normal shape and the cartilage is normally thick and healthy, it would take an abnormally heavy weight to change those joints. Thus a constitutional factor already comes into the picture, *viz.*, joints of congenitally other than normal shape and cartilage of inferior quality. It should be added that a normal joint can be subjected to strain of constitutional origin, as in the case of a patient who, on account of his constitution, moves in an abnormal manner or has an abnormal posture.

evident rheumatism or heavy excess pressure resulting from a malformation elsewhere in the body. Into this category come those individuals in whom Schmorl, Van der Meer, Veil and many others report the incidence of arthrosis at a very youthful age, such as children from 6 to 12 years old, etc. An endogenous constitutional factor was suspected in those persons who, without any demonstrable exogenous incidents, began to suffer in middle age from the signs and symptoms in the joints of a "worn-out" vertebral column which are usually associated with people of more advanced age. This was frequently seen to occur in the climacteric. In pre-senility there are gradations to senility when the pathology is less evident. The emphasis laid in the literature upon the occurrence of these premature signs of senility in middle age cannot escape notice. Osteoporosis and changes in the cartilage form the basis of the pathological changes. Otherwise, through constitutionally favoured diseases, at that age hypersensitivity may arise, inducing recurrent infectious conditions with the same secondary changes in the joints.

It is likewise astonishing to find the frequent occurrence of spondylarthrosis deformans in young people recorded in the literature. According to Schmorl the peak is in the period of 40 to 49. Van Assen saw that the facets of the joints were worn down in 60% of healthy persons between the ages of 20 and 40 and that this was so in 100% of the healthy individuals of over 50. It emerges from Van der Meer's material that the incidence of arthrosis deformans is just as common between 30 and 40 years of age as between 50 and 60, though there is definitely a qualitative difference. In these periods of life there are young people with old cartilage and old people with young cartilage (Burckhardt) and, if it be asked why this is so, the answer is merely that it is a matter of constitution.

Summarising what has hitherto been said as to the aetiology of arthrosis, we find that:

a. The vast majority of authors consider *strain to be the principal cause of arthrosis deformans*, endogenous factors being in part responsible for the intensity with which and the period at which this malady occurs.

b. A smaller number of authors consider arthrosis to be *secondary to arthritis suffered by the patient.*

c. The frequent incidence at advanced age is ascribed to prolonged exposure to strain and, to less extent, to *changes in the bone tissue* itself (osteoporosis, etc.) deriving from a hormonal discrepancy, vitamin deficiencies and errors of diet.

d. Only a few authors ascribe these symptoms of senility to *chronic faulty diet* (calcium deficiency); they consider that the period of life at which *premature*

secondary changes in the joint may arise. Summed up more briefly, rheumatism is, if not synonymous with arthrosis, then at least its precursor. In his view there is an intimate connection between chronic rheumatism (especially primary chronic polyarthritis) and arthrosis. He ends by describing arthrosis as a disease which should be defined, not so much as a local orthopaedic disorder, as a clinical picture in which reactions reminiscent of inflammation between the joints and adjacent structures are the central feature. Both aetiologically and pathogenically, a series of *conditionally* active factors are involved, such as infection, constitution, age, endocrine influences and, finally, mechanical effects of a static and traumatic nature. Meteorological irritants, whether or not combined with an allergic mechanism, could start off the inflammation-like reactions. Although Van der Meer's views are cited several times in the Dutch literature, one does not generally come across this one-sided standpoint in the international literature.

Van Assen and Lubberhuizen repeatedly refer to excessive strain upon the vertebral column as causing spondylarthrosis, an opinion also held by Chapchal, Epstein, Lewin, Schinz, Schmorl, etc. Van Assen is underlining the idea of secondary spondylarthrosis, or arthrosis deformans, of the vertebral column when he points out that a malformed joint does not make itself felt in youth, but does do so in the third or fourth decade. He ascribes this to adverse static relations.

Several authors assume that constitutional factors are responsible for spondylarthrosis Schmorl (p. 179) says· "Der konstitutionellen Veranlagung ist die grossere Bedeutung zuzumessen". In suggesting criteria by which to judge whether certain disorders are consequent upon accidents or not, Vogelensang states it as his opinion that a primary arthrosis deformans develops as the result of endogenous disturbances and a hereditary arthritic constitution As such, it would be a disease of the system, attacking several joints, and, as a rule, does not result from an accident or strain! Bauer and Bennett hold that the "quality of the cartilage that one inherits" is, in addition to other factors, decisive for the age at which the changes begin to take place, but also for the rate at which they progress

Spondylarthrosis used to be regarded mainly as a disease of old age. Glimpses of the endogenous character of the condition were had when it was realised that it could also attack young people When the vertebral joints of a person of middle age were observed to present the aspect of those of a greybeard, endogenous factors which had brought this condition about prematurely were held to be responsible.

To be able to appraise such a condition, it was of course necessary to exclude those cases which derive from a clearly exogenous basis, like a patent trauma,

evident rheumatism or heavy excess pressure resulting from a malformation elsewhere in the body. Into this category come those individuals in whom Schmorl, Van der Meer, Veil and many others report the incidence of arthrosis at a very youthful age, such as children from 6 to 12 years old, etc. An endogenous constitutional factor was suspected in those persons who, without any demonstrable exogenous incidents, began to suffer in middle age from the signs and symptoms in the joints of a "worn-out" vertebral column which are usually associated with people of more advanced age. This was frequently seen to occur in the climacteric. In pre-senility there are gradations to senility when the pathology is less evident. The emphasis laid in the literature upon the occurrence of these premature signs of senility in middle age cannot escape notice. Osteoporosis and changes in the cartilage form the basis of the pathological changes. Otherwise, through constitutionally favoured diseases, at that age hypersensitivity may arise, inducing recurrent infectious conditions with the same secondary changes in the joints.

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d. Only a few authors ascribe these symptoms of senility to *chronic faulty diet* (calcium deficiency); they consider that the period of life at which *premature*

symptoms of old age occur is determined by the gross or minor deficiencies which these faulty diets have brought about.

e. Arthrosis – often without symptoms – appears to occur to an alarming extent in young and middle-aged people. It is clear from the literature that the premature incidence of (arthrosis as) a symptom of old age is almost unanimously considered to be *determined endogenously*, *i.e.*, through a hormonal discrepancy symptoms of senility are liable to occur in pre-senility, during the climacteric or even before then. As old-age osteoporosis has something to do with the genesis of arthrosis, reference may be made to page 83 for the relation between senile, pre-senile and climacteric involutional osteoporosis.

In the literature no doubt is expressed at all as to whether an affection of the small vertebral joints can produce *back pain.* This is so unanimously taken for granted that we do not require to discuss it further here. What does need to be discussed, however, is the mechanism by which this pain is eventually produced. We have to make a distinction between low back pain and radicular pain. Opinions are also agreed on the cause of low back pain through derangements of the vertebral joints. As has been described by Armstrong, Wiberg and others, the interior of the spinal canal is nourished by nerve fibres which are part of the nervi sinuvertebrales and also supply the joints. In the presence of spondylarthrosis, changes take place in the synovia, cartilage, periosteum, the capsule of the joint and surrounding ligaments. The secondary changes produce pain in the same way as any joint disorder is presumed to do. Armstrong lays particular stress on the oedematous swelling of capsules and ligaments.

Several authors, if not all, refer to *radicular pain* as accompanying spondylarthrosis. One or two state that there may be monoradicular pain, though they do not explain why the referred pain occurs in the innervation region of one root In Armstrong's opinion (p. 118) it is strange that only one root should sometimes be irritated in a disorder as diffuse as spondylarthrosis. He does not suggest a *predisposing factor*, such as we envisage, in explanation, through which the space at the site is already smaller than it was before. He is probably more preoccupied with *concomitant influences*, such as a small hernia nuclei pulposi, lipping and so forth. Other authors mention *local spondylarthrosis*, hence contrary to Armstrong, who regarded the ailment as a generalised one. Van der Meer (p. 93) does not offer a clear explanation for these purely local changes of the joint, but he does cite other authors who ascribe this limited spreading of the process to insufficient arterialisation of certain joints or parts of a joint.

Schnitker and Curtzwiller (1957, p 121) speak of "hypertrophic osteosclerosis with bony spur formation of the lumbar spine". They assemble

several arguments to explain monoradicular pain, *viz.*, in addition to the formation of a bony spur, the laminae have thickened (restriction of space), the vascular system is plethoric and the ligamentum flavum locally hypertrophic. The bone is soft. The facets have become uneven through degeneration. The aetiology is thought to be probably traumatic; but Epstein (p. 177) assumes that the condition was preceded by arthritis. "This bony spur formation was already mentioned in 1949 by Hadley, speaking of so-called osteoarthritis: a non-inflammatory degenerative process".

The following authors seek the cause of root involvement in spondylarthrosis in *inflammation*, *oedema* or compression of the root. Bradford and Spurling (p. 85), as well as Van der Meer, emphasize inflammation of the root. Armstrong, Badgley, Milton and Love mention oedematous swelling of the root itself as one of the causes. The same authors do not omit to allow for the fact that an oedematous root is all the more likely to have insufficient clearance if, in addition, the intervertebral foramen should have been narrowed by the changes in the joint.

Ayers, Ghormley, Hadley, Putti, Schmorl and Zukschwerdt are mainly concerned with the *narrowing of the intervertebral canal*, adding that oedema of the root further aggravates the results of this narrowing. Hadley describes this postarthrotic narrowing of the foramen in a long article dealing with the subject.

In our opinion, what now follows is highly important. These authors stress the congenital shape of the joint. Ayers and Putti state that the joint forms $\frac{1}{4}$ to $\frac{1}{3}$ of the border of the whole intervertebral foramen. Accordingly, to their way of thinking, disorders of a joint are important factors in the narrowing of the intervertebral canal. The more evidently the shape of the joint congenitally departs from the normal, the more readily will the foramen be narrowed; and this all the more so if the congenitally abnormal shape of the joint is of a kind to favour spondylarthrosis. In the literature, the narrowing of the foramen by part of the surface of the joint is denoted as "facet syndrome" (Bradford and Spurling, Danforth, Putti, Wilson). Ghormley and also Love, in particular, mention it at length. He realised that not only do the joint facets constitute part of the foramen, but also partly cover the roots in their course. He therefore considers decompressive laminectomy as not going far enough in some cases unless those protruding parts of the joint are removed. The term "facetectomy" has become current in the literature and several surgeons perform that operation. We have already alluded to congenital changes in the shape of the joints, while the above writers generally mean the normal structure of the vertebral joint and only refer to the congenitally abnormal structure as an exception. We shall mention these exceptions presently.

Some authors, like Armstrong, consider the *clinical picture of spondylarthrosis* to be so typical that it can be diagnosed on the basis of the clinical history itself. And it is a fact that exacerbation of symptoms after rest, especially on rising in the morning, and their subsidence after a certain amount of movement, are almost pathognomonic for this malady (see page 258).

Van Assen (Sleeswijk, p. 553), however, has drawn up an entirely differential diagnosis of spondylarthrosis. Let us here mention: very many diseases of the internal organs, such as cancer of the stomach, gastric ulcer, anaemia and lung tuberculosis. Furthermore, spondylitis tuberculosa must be taken into account, as well as senile osteomalacia and osteoporosis, Bechterew's disease, Paget's disease, tumours, congenital vertebral deformities, static complaints (deriving from weakness of the muscles and ligaments) and arthropathy with tabes. The majority of the back complaints should in such cases, especially in association with internal diseases, be regarded as referred pain. We shall discuss this phenomenon separately.

It could be argued against this that spondylarthrosis deformans is in any case recognisable by X-ray, making differential diagnosis comparatively easy. But Lubbershuizen (Sleeswijk, p. 628) makes it clear that this is not quite true. In certain cases the patient suffers most from the capsule and surrounding muscles. Hence the appearance or otherwise of abnormalities in the radiographs should certainly not be taken as decisive. There are patients with very few symptoms whose radiographs show up serious abnormalities, just as there are other patients with many complaints with little to show on the radiographs. Krayenbuhl comes to more or less the same conclusion in a description of the disc syndrome. He begins by explaining that the radiographically apparent narrowing of an intervertebral disc need not necessarily provide evidence of a disc lesion. It does in only about 50% of cases. In 15% to 20% of hernia patients he saw signs of lumbar spondylosis deformans. He attaches little value to the radiographical detection of spondylosis, as combinations of a hernia plus this disorder do not occur in clear association with each other. The underlying theme of this report is, therefore, that radiological evidence of spondylosis and spondylarthrosis does not provide certain grounds for ascribing the symptoms to these disorders, but that the possibility of the existence of a disc lesion should be borne in mind.

Hadley, Harris, MacNab, Milton and Schmorl see a clear *relation between degeneration of the disc and the genesis of spondylarthrosis*. In their opinion all manifestations of a disc degeneration, especially narrowing of the disc, set changes in the vertebral joints in motion and, they say, *secondary* spondylarthrosis results from these.

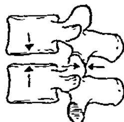
After this survey we are perhaps better able to understand that a causal relation is recognised between the occurrence of *congenital vertebral joint deformities* – whether or not endogenously caused – and the symptoms. When hernia nuclei pulposi was still unknown as a clinical picture, congenital changes in the vertebral joints received an integrating meaning in the occurrence of back pain. These malformations – if congenital disorders of the vertebral joints can be so called – occur so often that one begins to wonder whether any particular shape can legitimately be called normal. After studying 3000 radiographs, Brailsford found that in 57% the lumbo-sacral joint facets were inclined in a backward direction, in 12% inwards and in 31% were asymmetrical or stood in an intermediate position. Pheasant and Swenson, quoted by Southworth, point out that the joint facets which appear to lie in a certain plane in the radiograph are found on dissection not to be as flat as they appeared. Every one of them displayed several curves of a given segment of such a joint facet. Southworth then proceeds to give the result of an investigation in which the L.4/L.5 and L.5/S.1 intervertebral joints were found to be symmetrical on both sides in 63.6% cases, while in the remaining 36.4% there was asymmetry, either at one level or at the other.

After making some anatomical study, Putti is convinced that the joint facets as a rule assume a certain shape varying around an equilibrium, but that the joint surfaces are flat and some asymmetrical. These anomalies occur predominantly in the L.5/S.1 intervertebral joints. He calls this condition an "anomaly of articular tropism". The anomalies are often seen in combination with transitional vertebrae and spina bifida. Milton and Zukschwerdt give a very similar description. For fuller particulars see Hagelstam (p. 110).

How are we to suppose that a congenital joint anomaly produces pain? It is a fact of practical experience that it causes the patients no discomfort up to middle age. Consequently, the congenitally abnormal shape *itself* does not produce pain. Hence some mechanism must come into play to start off the complaints and the obvious conclusion in the case of joints is that this must be a mechanism of movement. Even if the joints are normal, too much pressure is liable to induce secondary changes as described in the genesis of spondylarthrosis. Where there are vertebral joint anomalies, a physiological use of the joint might be enough to bring about these secondary changes. So that this may be clearly understood, we shall briefly describe the *function of the vertebral joints*. They serve to absorb the static pressure and to enable the vertebrae to move in relation to each other.

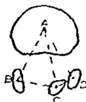
The weight of the body is best absorbed by the broadest possible horizontal surface and this is provided by the intervertebral disc between two vertebral

bodies. Smaller joint surfaces, gliding vertically side by side, suffice to conduct movements (hence not to absorb vertical pressure), or may, indeed, do so more effectively. For this we have the small vertebral joints, which serve primarily for allowing, directing and steering forward, backward and lateral movements. Now, if one of these small vertebral joints, the function of which is, fundamentally, to guide, is built in such a way that it tends to absorb pressure, its liability to wear down is greatly heightened. This happens when some small, usually lumbo-sacral, joints are directed more horizontally than vertically. In a certain posture, asymmetrical small vertebral joints will not be called upon to cope with more than a physiological burden. It is quite conceivable, however, that, owing to asymmetry, or if the joints are not entirely congruous, a certain posture will cause rotation. Thus, even a physiological movement may bring to bear excessive local pressure upon an incongruously placed (vertebral) joint, with increased wear and tear as the result.



The function of the joint of a vertebral body as a whole can be represented as the simplest form, in mechanics, of placing an object in stable balance. This is obtained by allowing it to rest on three points of support. It is not a mere matter of chance that this principle of the "tripod" is to be found both in nature and underlying the most primitive products of human ingenuity. As soon as 4 points of support are resorted to, the risk is incurred of one of the four not lying in one plane with the others and, therefore, of one point not being utilised. There are then two stable balances, each with three points of support, which may merge across an unstable intermediate position with two points of support. (Table with one leg longer or shorter than the others.) Constant oscillation from one stable balance to the other will bring greater pressure to bear on the two firm points of support. In the event of some mechanism being brought into action in an endeavour, none the less, to make use of all four supports (heavy weight on the table), torsion would ensue; the two longer legs of the table are dislocated. (If, because one leg is longer, the four points of support were not to lie in the same plane, it would be this abnormally long leg that would be dislocated under extra pressure.)





Rotation is a common phenomenon in physiology and can be initiated by a similar mechanism. If the setting of one of the small vertebral joints is not

exactly right, a fourth point of support comes into the picture. In some particular posture, only three supports, A, B and C, or A, C and D, are used. Oscillation around the equilibrium A-C brings greater pressure to bear on the intervertebral disc and part of the abnormal joint. Through strain, a pair of joints may be twisted.

It is clear that torsion brings heavy pressure to bear on the structures, mainly upon one part of this abnormal joint. Accordingly, we should interpret the dysfunction of the small vertebral joints as a disruption, due to overloading, of a function which does not rightly belong to the joint. As the congenitally abnormal vertebral joints decline in flexibility and resilience with advancing age, they are no longer up to the extra tasks imposed upon them and an abnormally vigorous process of wear sets in. In the same way, spondylarthrosis, or possibly only oedema, etc., will more readily develop in the presence of congenital malformations of the vertebral body under otherwise physiological conditions. We are thus able to postulate a relationship between the occurrence of these abnormal joints, the absence of symptoms during a certain period of life and the degenerative, proliferating alteration of these small joints.

When Southworth states, therefore, that he found asymmetry of the small L.IV/L.V and L.V/S.I joints in about 25% of a group of soldiers who had *never* suffered from back complaints, the fact that *none* among this group he had examined *were older than 40* fits perfectly into the pattern outlined above. It is entirely in accordance with our view that these persons had not yet come to develop arthrosis, arthritis, scoliosis or some other postural abnormality due to extra pressure upon congenitally malformed joints.

The occurrence of back trouble as a secondary result of congenital anomalies has thus become comprehensible; radicular pain is liable to be experienced when the abrasive processes have brought about arthrosis and spondylosis.

The foregoing can be summarised briefly in tabular form.

According to the literature, changes in the vertebral joints presenting the aspect of spondylarthrosis may take place as follows:

Trauma: fracture, luxation.

Inflammation oedema.

Rheumatism cold, damp.

Extra pressure on the normal joint

Physiological or exaggerated pressure on a joint *weakened* by·

a injury or inflammation

b. cartilage of inferior quality

c a congenital anomaly of shape.

The following *endogenous* factors are involved:

1 Primarily constitutional through deformity of the joint or inferiority of cartilage.

2. Secondarily constitutional when an over-sensitiveness to infection or the existence of allergic conditions is endogenous.
3. Secondarily constitutional when, through endogenous factors, posture and motoricity are or become pathological to such a degree that in that particular individual they cause undue pressure to bear upon the joints.

It seems evident from the above particulars collected from the literature that the vast majority of authors consider the occurrence of changes in the joints at a given time to be, partly at any rate and possibly largely, determined endogenously.

We therefore take spondylarthrosis to be:

a result of faulty use of the body;

a form of reaction to a number of maladies.

Endogenous factors are also responsible in part for the extensiveness of the disease and for the period at which it develops or manifests itself clinically.

Chiropractors, in particular, call attention to *luxations* and *pseudo-luxations* of the small intervertebral joints. It is noticeable that in the literature this abnormality is referred to in vague terms (Veraguth, p. 158). Publications dealing with the manual reposition of the lumbar spinal column are more explicit. One of the difficulties in the way of establishing the luxation of lumbar small vertebral joints demonstrably is said to be the fact that these minor dislocations do not appear on radiographs (Quetsch, p. 11).

The possibility of a dislocation is considered on the following grounds:

1. Luxations and subluxations of the cervico-thoracic vertebral column do appear on radiographs.
2. The function of the small lumbar vertebral joints is compared with that of other, larger joints, which are certainly known to be subject to luxation. There are undoubtedly striking points of similarity, such as the acute onset of the symptoms brought on by an awkward movement (the "unco-ordinated" movement, as Glorieux calls it)
3. The chiropractor's claim that the symptoms sometimes cease abruptly after a manual reposition also deserves mention This is ascribed to the redress of a luxation. It is alleged that a click can be heard, sometimes even at a distance, or, in other cases, with a stethoscope, as the dislocated joint jumps back into place.

Zukschwerdt (p 158 and elsewhere) expatiates on these arguments and mentions the names of several authors who support them. Yet there are several objections that could be raised against this very simple interpretation, *viz.*,

(a) this clicking can be heard accompanying the movement of many joints, without symptoms and without previous luxation of the joint;

(b) the successful result of manual reposition is by no means as common as the chiropractor makes it out to be Moreover, syndromes occur following a course

identical with the syndrome associated with a condition said by the chiropractor to be a luxation or subluxation, yet fail to yield to attempted reposition.

If luxation occurs – and we are quite sure that it does – this phenomenon cannot be held as generally responsible for pain in the back as is done in this one-sided literature.

4. The opinion that a small vertebral joint – more precisely, in this context, a lumbar vertebral joint – can be dislocated by an injury or an unco-ordinated movement, is also founded on post mortem experience (Harris and MacNab). At autopsy, haematoma was found in the small vertebral joint of persons who had held the back painfully fixed after a traumatic lesion of the vertebral column (Zukschwerdt).

5. At operations, but also at autopsy, small fragments of articular facets were detected in the vertebral joints This is the experience of several authors (Harris and MacNab, Zukschwerdt).

6. To our mind, however, the strongest argument in favour of the possible occurrence of luxations in all joints, including the small joints of the lumbar spine, is the anatomically demonstrable existence of a mechanism in the joint which can have but one purpose, and that is to prevent dislocation. With full documentation and mention of the names of authors who have experienced the same (Brocher, Sante, Töndury, Veraguth and Von Schminck), Zukschwerdt describes the occurrence of small *intra-articular menisci* in *all* joints of the spinal column. Seen under the microscope, this platelet, only a few millimetres thick, is of the same composition as, for instance, the meniscus in the knee joint. Following upon this, he found a *fold of tissue*, arising from the joint capsule, which spread between the two surfaces of the joint. In addition to these two tissues, he describes a small *lump of fat*, which we, too, have seen repeatedly after opening the joint.

The function of these three tissues is comparable with that of the similar structures in the big joints, *viz.*, to help the joints to slide and guide.

When, through an abnormal movement, the segments of the joints slide too far, so that subluxation threatens, the fold of tissue proceeding from the capsule will remain where it was between the two joint facets. Upon cessation of the abnormal movement, the dislocated part of the joint can slip back over this fold and, probably, over the meniscus between the joints to its normal position. Thus we believe that the anatomically demonstrable existence of a mechanism designed to prevent luxation or subluxation offers indirect proof that such dislocations of a joint can occasionally occur We have not been able to make out from Zukschwerdt's monograph, in which he deals extensively with the joints, whether he believes that luxations or subluxations of a small joint do or do not occur and lead to locking of the back. It might be inferred that he does not rule out subluxation, but ascribes the symptoms in the back which block movement to pinching of these menisci, folds of tissue or lumps of fat.

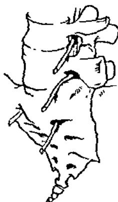




The sacro-iliac joints

What has been said in the preceding paragraphs applies, *mutatis mutandis*, to the joints which attach the sacrum to the pelvis. Opinions differ as to whether this is a true or a pseudo-joint. That there is some mobility of the sacrum in relation to the pelvis had been shown by X-ray and is all the more evident if radiographs are taken at fairly long intervals with conditions changing in the interim, such as in the last months of pregnancy and a few months after labour. Rudimentary vestiges of a joint are seen in specimens and the transition between the sacrum and pelvis appears to be more ankylotic. The interspace is bridged by many strong ligaments. The facets of pseudo-joints are erratic in shape and also vary considerably in diameter. In some places the joint seems to be 3 to 4 cm thick; in others it is no more than ½ to 1 cm. This is important, because when injections are given in the sacro-iliac joint, the point of the needle may emerge at the anterior of the joint, with possible damage to, or anaesthetisation of, the L.4 and L.5 roots. In that event, the patient often complains of a numb feeling in the leg. Practitioners should, therefore, be on their guard never to treat the sacro-iliac joint with alcohol or other substances injurious to the nerves. It is pointed out that the joint is surrounded by exceedingly strong muscles which assist it in its functions. On the other hand, the joint can be injured by powerful contractions of the muscles (Veraguth, p. 151). This may happen the more readily in that this is often a pseudo-joint which gives only partly to forces exerted upon it. The result may well be stretching of the ligaments and haematoma (Jappert). The local lesions are liable to become infected; and it is likewise possible that a bursa becomes oedematous or undergoes other changes, due to excessive pressure on the joint or ligaments, and produces secondary symptoms as of inflammation.

Freiberg, Vinke, Williams and many others, quoted by Bradford and Spurling (p. 114), declare that a lesion of the sacro-iliac joint is rare. According to Chandler, operative ankylosis does not invariably produce satisfactory results. It is said that one should not be too prone to seek the cause of low back pain in disorders of this joint. On the other hand, the same writers point out, as does also Armstrong (p. 114), that radicular pain may be produced by irritation of the fourth and fifth lumbar roots at the anterior surface of the joint. This, according to Armstrong, is more particularly the case when a large transverse process of a sacralised fifth lumbar vertebra causes a pseudarthrosis at the site of the sacro-iliac joint, secondary inflammation of this joint and



bursitis cause radicular pain, as Freiburg and Vinke also supposed. Willis (1937, p. 745), again, definitely associates disorders of the sacro-iliac joint with low back pain. Van Assen (Sleeswijk, p. 558) is most insistent that persons with postural anomalies should wear a corset, because the sacro-iliac joint is subjected to abnormal pressure; this is certainly so in the case of obesity, when the muscles are unable to furnish the support necessary to maintain a physiological posture. Since there is also such a thing as arthrosis deformans of the sacro-iliac joint, practitioners are warned of the danger of prescribing absolute rest, in consequence of which the muscles may weaken further and the arthrosis worsen.

Can the affections of the sacro-iliac joint be differentiated as the results of exogenous and endogenous factors? Exogenous factors, taking the form of injury and inflammation, have been mentioned and we leave local malformations due to diseases of the bone (osteomalacia, osteoporosis) and tumours out of consideration. Either typical endogenous disorganisation of the sacro-iliac joint is not indicated in the literature, or else it is in the same way as was done for the vertebral joints. Van Assen puts it like this: "Congenital malformations, and those developed in youth, predispose the subject locally to arthrosis deformans".

The abnormalities observed in the presence of *spondylitis ankylopoetica* (Bechterew's disease) are of mixed endogenous and exogenous character. This disease has been described in monographs and is, moreover, dealt with in several textbooks; we would specially mention the monographs of Aufdermauer, Boni and Kaganas. In view of the importance of the immature stage of Bechterew's disease to its differential diagnosis from other disorders causing back pain, we shall here dwell on some of the outstanding aspects of this condition. Its aetiology is not clear; endocrine disturbances do take place; it is thought to be hereditary (Stecher, p. 44). The disease begins to manifest itself early in life, usually at the age of 18 to 20; shortly after, the patient has back trouble and, in the untreatable forms, marked deformities appear at middle age. Low back pain may be an early symptom and occasionally simulates the low back pain associated with a disc lesion. At a later stage the patient complains of pain between the shoulders, sometimes radiating to both arms, with tremors and clear signs of neural loss. This is ascribed to compression of cervical roots. Radicular pain in the leg may accompany the middle-age low back pain as the result of changes in, *i.e.*, narrowing of, the intervertebral foramina. Hence, broadly speaking, the symptoms outlined are sometimes reminiscent of those of hernia nuclei pulposi. But the thoracic kyphosis and the typical shuffling gait are indicative of Bechterew's disease and the next step is to

screen the whole spinal column. The radiographs will then show the familiar bamboo-like changes in the cervico-thoracic spine; the ligaments are invaded by bone tissue or cartilaginous tissue, so that it looks as though wax had been poured over the column.

The sacro-iliac joints are changed (ankylosis) at the onset of Bechterew's disease and these changes may be the first of all the symptoms to make the physician suspect this condition. In advanced stages, these joints are barely visible. This initial change in the sacro-iliac joints is a most valuable datum for early diagnosis, because it enables timely X-ray treatment to be tried out. The bone tissue at the anterior cervico-thoracic vertebral column and the diminishing intervertebral discs become fibrotic; ossification of the connective tissue leads to curvature of the spine until the patient is "bent double". The purpose of X-ray therapy is to soften these foci of tissue so that, by subsequent controlled exercises, the postural forward trend may be corrected.

The sedimentation rate of the erythrocytes is initially slightly raised, but may become very high later on. The detection of eosinophilia assists in the confirmation of the diagnosis.

The patients usually complain of recurrent iritis or iridocyclitis, for which no explanation is forthcoming in the literature so far. It is regarded as one argument, among others, in favour of allergic-endocrine disorganisation as the origin of the disease. Veraguth, p. 140, speaks of a special form of rheumatism.

In view of the fact that this disease of the system is probably hereditary (in male progeny) and that it so typically attacks at a certain age, opinion on the whole, as expressed in the literature, tends to characterise it as determined endogenously.

The Intervertebral Foramen

This term is generally used to convey – *pars pro toto* – the opening in the lateral wall of the spinal canal. Hadley calls it the cross-road between the mobile part of the vertebral column and the peripheral nerve system.

It is misleading to define this opening as no more than a space between two vertebrae; actually it consists of four contiguous parts, viz,

1. the approach to the foramen,
2. the entrance to the canal, *i e.*, the intervertebral foramen;
3. the intervertebral canal and
4. its exit.

The adjacent parts are: an intervertebral disc, two posterior sides of the

adjacent vertebral bodies, 2 pedicles, 2 articular processes with a joint in between and the joint capsule, the ligamentum flavum and other ligaments. The roots pass through the openings, or the ganglia are situated there, merging into the spinal nerves; there are, moreover, arterial and venous vessels and fibrous connective and fat tissue.

It should be remarked in passing that we are here dealing more especially with *low* back pain and are therefore mainly concerned with the foramina for the L.4, L.5 and S.1 roots. These three foramina are very dissimilar.

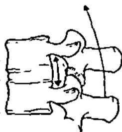
Our comparative ignorance of the topography and pathology of the intervertebral foramen *in vivo* is due to the fact that this region is difficult of access. Conservative treatment of disorders localised at this site depends largely on guess-work; at operation the field is found to be very difficult of access, while it is hardly feasible to get the tridimensional aspect reproduced radiographically.

It struck us during our study of the literature that neither the photography nor drawings made from life provide a clear picture of the *anatomy and topography of the intervertebral canal*. We believe the functioning of this system is brought out most clearly by diagrammatic representation in the simplest possible form, which is our reason for limiting ourselves to this. Even the sharply focussed exposure taken at very close quarters and magnified many times is disappointing, both because it fails to give a comprehensive view and because the intermingling of the tissues makes them nearly indistinguishable one from the other.

The intervertebral canal system has a threefold *function, viz.*,

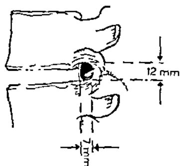
1. Primarily this space serves to afford a joint room to move and its function is therefore intimately connected with that of a joint.
2. It also provides passage to nerve and vascular tissue.
3. It provides shelter for the spinal ganglion.

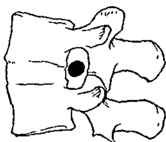
Re 1. The mobility of the vertebrae in relation to each other depends on the cushioning function of the disc and the conducting function of the small vertebral joints. When the spine is bent backwards, the dorsal inferior articular process glides over the ventral superior articular process and this reduces the size of the bony border of the canal. The changing of the dimensions upon movement can be verified radiologically. With a sideways bending the joints rotate and glide, through which a virtually identical mechanism operates. Upon forward flexion the foramen seems to become larger, because the articular facets shift apart. They are, however, checked by the capsule and ligaments, which



then tighten more and thus form the arc of a segment of a circle and in this way again reduce the size of the foramen.

On injecting saline into the joint capsule, Magnuson noted that the foramen narrowed ventro-dorsally by 2 mm (which is more than 25 %), incidentally thus proving that the ligaments are also capable of changing the dimensions of the canal. They also apparently contribute to the reduction of its size brought about by dorsiflexion, for the bony margins then approximate each other; the stretched ligaments are too long and become thicker, as is seen to happen in any more or less elastic tissue.

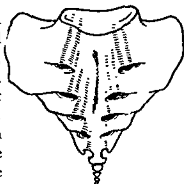




Magnuson gives a little more than 7 mm in the dorso-ventral direction and 12 mm lengthwise as the average diameter of a lumbar intervertebral foramen. He says that the average diameter of a lumbar ganglion is 7 mm, *i.e.*, only fractions of a millimetre smaller than the space within which it lies. In view of the considerable individual disparities, we think it is rash, to say the least, to accept such averages. Admittedly, with this author's measurements in a given instance, it is clear that there is little spare room for the ganglion and that any minor adventitious limitation of space would hem it in. Several authors, however, describe a different proportion between the diameter of the ganglion and the foramen which appears to us to be more correct. Hadley estimates the ratio to be 1 to 5 for the cranio-caudal dimensions, but it is fairly certain that he must have meant the ratio of the ganglion to the bony intervertebral space as is seen in photographs, and then in its greatest cranio-caudal dimension. Garland, quoted by Hadley, gives it as his opinion that this is so only in the cervico-thoracic vertebral column and that the lumbar ganglion almost completely fills up the intervertebral foramen. The impression received from operative findings is that, in the dorso-ventral direction, the ganglion is very little smaller than the foramen, but that in the cranio-caudal direction it takes up one-third of the space, the remainder of the canal being filled with loosely fibrous tissue and a little fat. The ganglion is thus well cushioned in a fairly insulated space, protected from external violence. As long as pathological processes do not change the reciprocal proportions, the canal provides ideal shelter. The same applies, *mutatis mutandis*, to the nerve-

root and the spinal nerve. These, too, have a definite size, adapted to that of the canal. Other authors, including Lewin and Schmorl, believe that the foramen adapts itself to the dimensions of the nervous system, Schmorl going even further by assuming that the dimensions of the canal are dependent upon its function of giving passage to the nerve tissue. He adduces as evidence (p. 29) the fact that, in the case of congenitally fused vertebrae, the intervertebral foramen is scarcely larger than is required to encompass the appropriate roots. He says that the opening, fixed in this case, does not call for any spare room to allow the root to pass just because the foramen does not change shape, *i.e.* become smaller, in response to movements. This is precisely the prevailing state of affairs where the S.1 root is concerned, which leaves the vertebral canal through an uninterrupted bony canal.

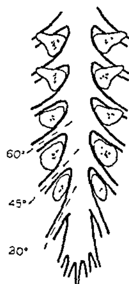
This is the proper place to describe the points of difference between the intervertebral canal and adjacent structures in respect of the S.1, L.5 and L.4 roots, thereby calling to mind the gradual transitions existing between the vertebrae themselves from the high cervical to the sacral. In the thoracic region, for instance, these gradations are so gradual that the anatomist is only able to name a given thoracic vertebra by approximation. This gradation in shape is still gradual in the high lumbar region, but is abrupt in the low lumbar. Finally, there is considerable difference between the L.V and S.I vertebrae. This abrupt change in type of vertebra from lumbar to sacral is responsible for the differences in shape and position as between the respective foramina. The various sacral vertebrae together form the sacrum as a single bony unit. Functionally, the intervertebral foramen under the first sacral vertebra is just an opening in the bone rather than an aperture between two vertebrae. This intervertebral canal has no moving skeletal parts either at its entrance or exit. Therefore, displacements of the joints accompanying movements, as in the other foramina, will be immaterial.



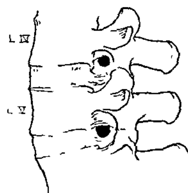
The *S.I intervertebral canal* is a flat-oval, rigid tube, the entrance and exit of which are covered by ligaments These only, through their function, might possibly narrow the entrance or exit, but, as they take part in movement in only a very minor degree, it is scarcely likely that they will do so. We have found by measurement that the length of the intervertebral canal for the S.1 root is liable to vary from ½ cm to 2 cm. On an average we found the length to be more than 1½ cm.

It should also be recalled that the course of the roots from the high cervical

to the low sacral region is entirely different, not to say contrary. We have on occasion seen the highest cervical roots running in an upward direction, much as someone might raise his arms sideways. On the whole, the cervical and thoracic roots run in a transverse direction and the angle at which they leave the dural sac becomes progressively smaller. The L.3 and L.4 roots stand at an angle of about 60° to the dural sac, the L.5 stands at 45°, the S.1 root at 30° or less, while the S.2 and other sacral roots run lengthwise almost parallel with the dural sac and filum terminale. It is because of this slanting course of the S.1 root that the canal through which this root has to emerge from the vertebral column is so long. For the same reason, the intervertebral canal in its entirety is somewhat longer between L.V and S.I (on an average 10 mm) than that between L.IV and L.V (4 to 6 mm on an average). This greater length of the L.V–S.I intervertebral canal is also ascribed to the peculiar shape of the superior articular process of the S.I vertebra. But it remains to be seen whether this latter be not the result of the former.



Besides being different in length, the *L.V–S.I canal* (for the L.5 root) is differently constructed from the canal for S.1, the former being composed of moving parts, *viz.*, joint, articular process and disc. Finally, it is due to the angle between L.V vertebra and the sacrum that the incisura superior of the S.I arch is smaller than that of the vertebrae situated higher up. Through this, the lower part of the bony L V–S.I intervertebral canal is distorted and smaller. This is seen at first glance on a lateral radiograph and is seen more clearly still on paramedian sections through the canal. The decrease in diameter of the intervertebral canal from the high lumbar to low lumbar region is generally referred to in the literature (including Bradford and Zukschwerdt). As a rule, therefore, the *canal between L.IV and L.V* is wider (7 × 19 mm on an average) than the L V–S.I canal (7 × 12 mm) (Larmon) It is shorter and, moreover, here the L.4 root passes through it in a slightly more transverse direction. The incisura vertebrae inferius of the L.IV vertebra is deeper, the disc lies lower in the plane of cross-section, with the joint result that the L.4 root

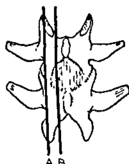


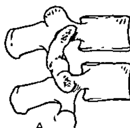
passes in a transverse direction high up through the canal without coming into contact with the intervertebral disc. Kirsch has described how the L.3 and L.4 roots have already left their intervertebral canal before they come into contact with the postero-lateral part of the intervertebral disc.

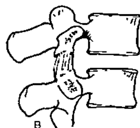
Some authors (Larmon, Lewin, Love, Zukschwerdt) view the intervertebral foramen as being delimited by the disc, pedicles, incisura vertebrae and ligamentum flavum. The same description is generally found among those authors who attach some value to a possible hypertrophy of the ligamentum flavum. The contradiction between this description, making a ligament the sole dorsal delimitation, and the radiographically observed fact that the opening is like a closed ring of bone, is only resolved by continuing to differentiate between the intervertebral canal just described and the entrance to it, *viz.*, the foramen; for, owing to the erratic contours of the space between the superposed arch and the medial delimitation of the vertebral joint, this foramen certainly is covered by the interposed ligamentum flavum. This is less markedly so at the entrance to the S.I canal and more clearly so at the entrance to the canal between L.V and L.IV.

Still further in the medial direction is that part which we call the approach to the foramen, entirely open towards the medial side, like a tube cut obliquely. The lateral border is provided by the incisura vertebrae, or the pedicle; part of the joint (facet, see Bradford and Spurling, Love and others) and the ligamentum flavum form the dorsal boundary. The median boundary is open: through it runs the root obliquely from the dural sac to the foramen. The term "sulcus lateralis" is given to the most lateral space of the foramen vertebrae, actually the angle between the arch, pedicle and posterior aspect of the vertebral body. This semi-canal could more aptly be called a groove or furrow. Little attention is paid to this in the literature. (Armstrong, p. 85, Cossa, p. 216, Spiegel, 1956, p. 192).

Chapchal, Love, Serpyener and others have, however, described a lamellar extension of the pedicles. This roof-like extension of the pedicles may re-







present a transition between the bony vertebral column and the intervertebral canal. The depth of this sulcus, *i.e.*, the extent and spread with which the arch here begins to form a canal for the root, depends on the shape of the arch. As this helps to determine the shape of the vertebral canal, we shall revert to it when describing the vertebral canal.

The pathology of the intervertebral canal

Although we do not really know what mechanism produces low back pain, the impression one receives is that this pain is in part ascribed to changes in the system of the intervertebral canal. Thus all disorders of the joints, such as arthritis, arthrosis, dislocations and so forth may well play their part in producing it. Other component parts of the foramen and canal are also subject to local changes, of which we may mention inflammation, tumours, fractures, lysis of the arch, and one is constantly finding that the authors hold oedema to be responsible for this low back pain (Cossa, Krischek, Thurel, Zukschwerdt and many others).

It has been pointed out (Hanraets, 1955) that, through the after-effects of spondylosis, posterior lipping, arthrosis, etc., a local restriction of space may ensue, owing to which parts of the canal or the foramen approximate each other. If these proliferations of bone occur on moving parts, these projections will be compelled by movements to rub over each other, while on the other hand static factors themselves probably induce local pain. The oedema presumed by others might be a secondary result. This condition is all the more likely to occur if the available space is narrower than the normal (constitutionally and sometimes radiographically demonstrable). The above complaints are due to limitation of the joint function of the intervertebral system.

It is difficult to see how radicular pain brought about by changes in the intervertebral canal can be caused otherwise than by *compression* of the nervous elements passing through it. Generally speaking, the following three conditions could be conducive to this:

1 The relationship between the available space and its contents may be upset if the traversing nerve tissue and the blood vessels are, congenitally or from whatever cause, larger than usual. This will be dealt with in a later chapter.



2. The available space itself may be constitutionally smaller. Examples would be lamellae-like extensions of the pedicles and the presumed hypertrophy of

the ligamentum flavum. Moreover, the shape of the foramen is liable to vary from individual to individual, sometimes being rounded oval and in other cases a flat oval for no demonstrably pathological reason. These limitations of space will have to be regarded as predisposing factors to absolute restriction



of space should other factors arise which further upset the normal relations between space and contents.

3. Space may become congested in a normally formed canal. Again it is necessary to make a distinction between absolute and relative restriction of space, the latter only predisposing to an absolute limitation if other factors should arise.

Re 3. A simple example of a relative limitation of space is that occurring in association with physiological movements as described above. These do narrow the canal, but there is still so much spare room left that the contents are unaffected. The prolonged persistence of a given posture, however, as with scoliosis, may permanently cause congestion. Finally, pathological movements will evoke this same mechanism again and again, thereby inducing secondary changes in the foramen and the canal which, as permanent predisposing factors, may eventually bring about absolute limitation of space if other factors accrue. It is difficult to assume, as is done repeatedly in the literature, that one ostensibly physiological movement is capable of inducing changes in a normal foramen, unless it also be assumed that there had already been a predisposing factor at work.

Here is a catalogue of disorders, collected from the literature, which can be taken as *acquired, persistent, predisposing factors* liable to cause relative or absolute restriction of space:

a. Residual conditions of fractured parts of the skeleton.

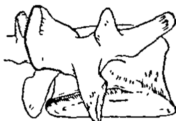
b. Luxations and haematomata.

c. Arthritis and arthrosis of the joint and associated changes in the joint capsule.

d. Spondylosis and posterior lipping (also see page 445, Part III). Schmorl points out that a distinction has to be made between spondylosis and posterior lipping, as the latter can occur without any signs of spondylosis elsewhere in the spine. The phenomenon is not invariably to be seen in radiographs, though

it can be established beyond doubt at operation and at autopsy. Schmorl thinks that one of its origins is the kyphosis of adolescence (p. 221), while elsewhere he suggests (p. 178) that it may develop secondarily through local pressure. This posterior lipping is reported very generally in the literature (Briggs, 1949, De Sèze, Hirsch, Krayenbuhl, Larmon, Thurel and many others). It is quite evident that this lipping is considered to be a contributory factor in the narrowing of the foramen and intervertebral canal; also that it may cause radicular pain (Hadley, 1949, p. 473, Schmorl and many others). Zukschwerdt goes as far as to assume that after the fourth decade all intervertebral foramina are changed by lipping, though, naturally, not invariably producing symptoms.

e. Posterior lipping has also been reported at the exit of the intervertebral canal. Larmon noticed at autopsy that the spinal nerve was there embedded in a deep sulcus and, furthermore, was covered by a firm ligament. The same thing is reported by Magnuson, who likewise observed an anomaly of the ligaments at the exit of the foramen, owing to which the nerve narrowly escaped being compressed. We consider this groove discovered by Larmon in the posterior aspect of the vertebral body to be the *result* of lipping. We have found a similar groove in the canal and also more often at the entrance to the L.V–S.I foramina than at L.IV–L.V.





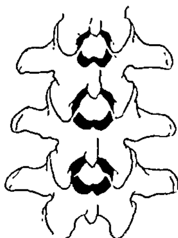
f. The posterior margin of the S.I vertebral body is physiologically arched, partly as the result of the angle formed by the vertebral column and the sacrum This arching is most pronounced at the site of the entrance of the S.1 root into the intervertebral canal. Of itself, it will not produce symptoms, but, if it is more than normally pronounced, it may constitute a predisposing factor to narrowing of the entrance to the canal (Hanraets, 1953). Melamed, on the other hand, described a concave posterior border of the first sacral segment, nevertheless, there can be some posterior lipping.



g. It has been noted at operation that the shape of the intervertebral foramen is sometimes a *flat* oval; it was observed in patients suspected of suffering from demineralisation of the skeleton (Hanraets).

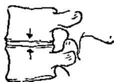
h. The intervertebral disc may bulge physiologically in a varying degree;

indeed, it is unusual for it to be entirely flat posteriorly. If this bulge is situated at the entrance to the foramen, the latter will be relatively narrowed. Clearly, the more this bulge tends to become a retropulsion, the more likely is it that this relative restriction of space will become an absolute one. To the extent that the intervertebral disc forms part of the intervertebral canal, the same mechanism prevails there. Owing to the fact that the borders of the canal are bony and do not, therefore, give way to the root, slight bulging of the disc in the canal will have graver consequences than outside it; likewise if the approach to the foramen crosses the intervertebral disc, when the root is covered on the dorso-lateral side by lamellae-like extensions of the pedicles and cannot get out of the way of the physiological, or slightly more than physiological bulge of the disc. As we see it, then, the diameter of the canal is reduced in size by other, quite physiological, accidentals (*e.g.* a physiological movement); owing to the slightly more than normal bulge of the disc at that moment, the root is compressed when this quite physiological movement is made and this causes pain or puts a check on the movement. This latter might account for the fact that the movements of some individuals are constitutionally checked, *i.e.*, it may explain why some particular person acquires a habit of moving stiffly. We even think it is plausible that acquired stiffness (pseudo-fixation on flexion, pseudo-positive Lasègue) of the elderly is attributable to a relative narrowing of the intervertebral foramina; atrophy of the muscles through inactivity and probably also weakening of the tendon reflexes would be secondary to this.



1. The intervertebral disc determines the distance between two vertebral bodies. If, in imagination, we take this disc away, the two vertebrae would







become contiguous and the superior and inferior articular processes would therefore slide over each other. This would reduce the size of the canal and foramen considerably. It would then depend on the position of the small

vertebral joints whether the displacement is vertical or whether the vertebral bodies also move in an antero-posterior direction in relation to each other; for, if this joint slants a little – as it usually does – the vertebral bodies will, as they approach each other, also be subject to slight retrolisthesis (pseudo-spondylolisthesis, according to Junghanns, p. 437). This further narrows the vertebral canal. It is repeatedly stated in the literature that the disc can be reduced in size without necessarily entailing a hernia nuclei pulposi; or it would be better to say that the collapse of a disc need not necessarily be the result of a nuclear retropulsion (Krayenbühl, Krischek, Lewin, Schmorl). It is ascribed to necrosis and degeneration of the intervertebral disc itself (concealed disc). Associated with this lesion, the vertebral bodies have been seen radiologically approximating each other, with slight displacement relative to each other, while radiographs likewise showed narrowing of the bony canal. The same is observed after radically "spooning" an intervertebral disc; and it is often seen on radiographs made after the removal of a hernia nuclei pulposi.

There are very good reasons for believing that the foramen is thereby so reduced in size that the root may be compressed; and it will the more readily be so if other factors, like posterior lipping, are also involved (Bradford and Spurling, Larmon, Lewin, Magnuson, Schmorl, Zukschwerdt and many others). It is, indeed, our considered opinion that failure to allow for this phenomenon in radical discectomy and to widen the canal and foramen as a precaution, is one of the causes of recurrent radicular pain.

j. A large amount of granulation tissue situated at the entrance to the inter-vertebral foramen has occasionally been noticed at re-operation (see page 624). Sometimes, too, the ligamentum flavum was distended, suggesting that hypertrophy of the ligament had been overlooked at the previous operation.

The growth of granulation tissue cannot be regarded as pathological. It is only to be expected that blood left behind will become fibrotic and lead to the development in some degree of granulation tissue. In our opinion, this tissue will only cause compression of the root if the space immediately in front of the intervertebral canal continued to be restricted after the previous operation. This might be so if a disc prolapse had not been removed in its entirety, but equally by posterior lipping or one of the other changes mentioned above. The coincidence of other predisposing factors with this non-pathological granulation tissue leads to absolute restriction of space. This can be prevented at operation by eliminating these predisposing factors as far as possible and by clearing the entrance to the foramen to make room for this granulation tissue (flattening the bony margins, Hanraets; foraminotomy, facetectomy,

Bradford and Spurling, Ghormley, Love; undermining foraminotomy, Briggs, 1945, p. 476).

We have not included in the preceding paragraphs those disorders which do not come within the functional province of the intervertebral canal system, by which we mean tumours and exceptional diseases of the bone, like Paget's disease and Bechterew's disease. In the latter it is said that the foramina may be reduced to one third of their diameter (Boni, Kaganos and Aufdermauer, Schmorl).

The Vertebral Canal

The ligamentum flavum

Like a band of connective tissue of varying thickness, the ligament is situated in the space between the arches. It is attached to the superior margin of the arch below, but runs on to the middle of the arch above, passing under it on its way. At operations we have sometimes seen the ligament running uninterruptedly under the arches without being fixed to them. Laterally it stretches up to the joint capsule and the intervertebral foramen. According to Armstrong and Zukschwerdt, it does not blend with the capsule of the joint. If that is so, it is difficult to see how the fold of tissue which extends between the small joint facets could arise from the ligamentum flavum, as described by the latter author. The lateral extension of this ligament is said to be partly attached to the extension of the arch and a part of the articular process and partly forms the dorso-medial boundary of the intervertebral foramen (Armstrong, Zukschwerdt). Although the ligament covers the whole length of the vertebral column, it is most strongly laid down in the lumbar region. Its function is to cover the spinal canal posteriorly. This is a protective function as far as absorbing pressure from a dorsal direction is concerned, but it can also be said to exert, in its covering function, counter-pressure against considerable difference in pressure in the epidural space. After surgical removal of the ligament, the dural sac is seen to swell under pressure-raising conditions; also, possibly, in the erect posture. The ligament further serves to check forward bending and it is more especially on account of this function that it is called the ligamentum flavum interarcualis.

Under the microscope, this yellow ligament is seen to contain elastic tissue cells. Macroscopically, adhesions have been observed, these probably being the result of infective diseases or traumatic lesions. Traces of traumata have also been found, taking the form of rents and old blood pigments. Calcification and local ossification have furthermore been reported, but we have been unable to discover any in a great many investigations. The thickness of the ligament varies considerably; Zukschwerdt mentions 7 mm, without hinting

that this is pathological. It is said to be far thicker medially than laterally. It depends upon the subjective attitude of the examiner whether a more than usual thickness of the ligament is attributed to hypertrophy; and it is primarily for the same reason that opinions as to the occurrence of hypertrophy are so divergent. Several authors suggest that the assumption of hypertrophy may possibly be a counsel of despair when no other cause for the symptoms can be found at operation. It is pertinent to this problem that no distinct hypertrophy was discovered during the examination of extensive *dissection* material (Bradford, Horwitz).

In their attitude to possible hypertrophy of the ligamentum flavum, the authors fall into three groups, *viz.*,

1. Those who accept hypertrophy and assume it to be a primary cause of symptoms (Girard, Lewin, Spurling, Veraguth).
2. Those who believe they have detected hypertrophy, but ascribe the symptoms to a combination of this and some other abnormality (Armstrong, Kirschek, Love, Roeder).
3. Those who do not consider hypertrophy of the ligamentum flavum to be probable (Bradford, quoted by Lewin, Cloward, Jackson, Krayenbuhl, Lindblom, Reischauer, Schmorl, Zukschwerdt).

The authors who do recognise hypertrophy, but only attach importance to it if it occurs in combination with some other cause of the symptoms, account for it by the already existing diminution of space as the result of thickening of the ligament, in which event an adventitious cause, such as a small disc lesion, might the more readily compress the root. Love (1939) states that he found a thickened ligament 155 times in a series of 175 patients operated upon for nuclear retropulsion. As against this, he only observed a hypertrophic ligament twelve times in 300 cases without an associated hernia nuclei pulposi. Krischek speaks of a "so-called" hypertrophy, for which he makes a temporary oedema responsible.

Those who firmly believe in the possible existence of hypertrophy of the ligament substantiate their belief, not only by what they have seen themselves, but also by the fact that, after extirpation of the "thickened" ligament, their patients ceased to present symptoms. We do not feel this last argument to be convincing, since the remission of the symptoms might equally well be attributed to other decompressive effects of a laminectomy.

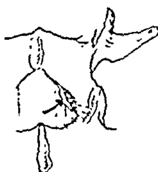
Our study of the literature, as summarised above, and our own experience of many laminectomies, in which a "hypertrophic" ligament was a very rare occurrence (twice in 2600), *lead us to the following conclusions:*

1. The ligament varies physiologically in thickness. A thick ligament is a

predisposing element in the narrowing of space; where it exists, therefore, other space-limiting factors have a better chance of making themselves felt.

2. We found that the ligament is thickest laterally if the patient has a wide vertebral canal; the free lateral margin at the site of the intervertebral foramen, especially, is then laid down thickly and also the insertion in the vicinity of the joints. It could therefore be said that people with a wide vertebral canal – about which we shall be speaking presently – constitutionally have a ligamentum flavum which, laterally, is more than usually hyperplastic.

3. Finally, there is the question of the elasticity of this tissue to be considered. If one cuts through this ligament at about 1 centimetre from an insertion, it retracts to form a thick lump of tissue close to this insertion. On account of its compact thickness, the thus remaining portion of the ligament looks as though it were hypertrophic, or hyperplastic.



We find support for this view of the matter in a statement made by Briggs (1949), who writes: "... after collapse of the intervertebral space, ... the disk bulges, and the ligamentum flavum thickens and bulges also".

This, to our mind, explains why so many surgeons were under the impression that the ligament was hypertrophied.

By contrast, anatomists find, on dissection, a rigid ligament which has largely lost its elasticity and, therefore, does not behave as described when cut.

It is when the vertebral canal is wide that the ligament tends most to bulge laterally. Hence there is an accumulation of elastic material which contracts when cut. Perhaps this also accounts for the fact that we thought we had found "hyperplasia" of the ligament in a wide vertebral canal lateralwards, at the site of the insertion and the intervertebral foramen; probably it was merely an accumulation of vestiges of the contracted ligament.

We believe, therefore, that our "hypertrophy", "hyperplasia" or lateral thickening of the ligament was brought about *at* the operation and was not even in part responsible for the pre-operative symptoms. That is not to say that to leave a plug of tissue in situ would not have pathological consequences; it might well take up so much room as to precipitate post-operative root adhesion

The posterior longitudinal ligament

This long, generally thinly laid down ligament runs inside the spinal canal at the back of the vertebral bodies. The collagen elastic connective tissues of which this ligament consists are grouped in thick bundles in the median line,

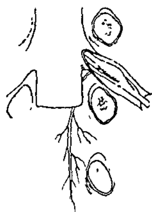


fanning out at the site of the disc right into the annulus fibrosus. The ligament is thickest medianly, varying by several millimetres (Zukschwerdt). Verbiest assumes that the thickness varies by 1 mm. (We measured up to as much as 3½ mm.) In the postero-lateral portion of the intervertebral disc the fibrous structure is thinner than at the site of the median bundles. It is precisely in this lateral position that nuclear retropulsion is most common.

Zukschwerdt says, indeed, that the lateral part of the disc is not covered by the posterior longitudinal ligament. He also points out that the dorsal lamellae of the annulus are the weakest by nature (Thurel). The predilection of hernia nuclei pulposi for the postero-lateral localisation is said to be due to these two facts. Albert-Lassierra reports the local absence altogether of the posterior longitudinal ligament in a number of laminectomies. We have never discovered this ourselves, but the thinness of the film covering a prolapse certainly is striking.

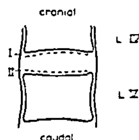
Nervi sinuvertebrales

Von Luschka was the first to describe these nerve fibres about one hundred years ago. Although they have since then been repeatedly mentioned in text-books and anatomical atlases, opinions as to their exact ramifications are anything but unanimous. It is assumed that these minute nerves innervate the posterior part of the disc, the interior of the spinal canal and a considerable portion of the joint. They are said to arise from the primary division (meningeal nerve) behind the spinal ganglion from the peripheral nerve (spinal nerve) and to re-enter the vertebral canal *via* the intervertebral foramen. Roofe (Krayenbuhl, p. 19) assumes that these bundles of nerves are distributed over the dorsal surface of the posterior longitudinal ligament and the annulus fibrosus, but do not innervate the cartilaginous end-plates The fibres are so thin (¼ to 1 mm) that they are not seen at operation. According to Veraguth (p. 83), they contain sympathetic elements. They are supposed to extend through the spinal canal in a common course with the vascular system. Von Luschka describes a simple extension of the nervous system in the vicinity of the foramen through which it enters the spinal canal Roofe lets the nerves descend along several segments of the vertebral canal. Wiberg and others supposed that



corresponding plexi of nerve fibre strands reached to beyond the median line (Armstrong, p. 13, Lewin, p. 710, Pedersen and co-workers, Spiegel 1957, p. 315, Zukschwerdt, p. 29).

To this we can add the following from our own experience. It has transpired that the excision of the caudal part of a lumbar disc is appreciably less painful than that of the cranial part. If the longitudinal ligament and the disc are cut first in plane I, then very little indication of pain is given upon subsequently cutting in plane II. Section in plane I after previous section in plane II produces unrelieved pain. This would seem to show that the distribution of sensory fibres in the lumbar vertebral column proceeds in a cranial to caudal direction (Roofe). Moreover, pain irritants in the lumbar canal appear to evoke sharper response in proportion as they are more closely proximate to the foramen. A further possible conclusion is that the cranial part of a disc is more effectively innervated (provided with pain-transmitting fibres) than the caudal part.



The sensitiveness of the ligaments, the annulus fibrosus and the joints is regarded as being a result of the innervation by these nerve bundles. From this it follows that, if these plexi could be disconnected in the foramen, the joints and the disc at the same level could be made insensible. This, in fact, is in accord with our experience. If, at a given level, the epidural space is voided of arteries and veins, the end-branches of these nervi sinuvertebrales will also no doubt be damaged, as they run together with the vascular system. We have noticed time and again that, when we started to manipulate in the epidural space, a patient being operated upon under a local anaesthetic would give signs of pain in response to mechanical stimulation of the disc and joint. When we had cleared the space, however, there were no further signs of pain. The same was found after manipulating around the ganglion; this is probably due to disconnection of the nervus sinuvertebralis in the foramen. We noted that patients whose vascular system had coagulated and then been extracted remained free from local back pain for about two to six months. It is reasonable to suppose that the interrupted nerve connections regenerate during the months succeeding the operation and that, in consequence, the local back pain returns in course of time.

The epidural space

The spinal canal contains fat tissue, connective tissue, vessels and small nerve stems, in addition to the dural space and the cauda equina.

The epidural space is narrow ventrally and wide dorsally, a contrast most marked in the lumbar region. Ventrally, the dural sac lies against the posterior walls of the vertebral bodies, separated from them by a plexus of veins and ligaments. Dorsal to it there is spare space of varying size, either filled with fat or not. This may be why pathological processes taking up room, like inflammation exudates and tumours, are often found on the dorsal side of the dural sac. Biemond and Prick, in referring to the epidural space, speak of an interdural, or intermeningeal space, thereby indicating that they consider the dura and the fibrous lining of the vertebral canal to be a visceral and parietal derivate, respectively, of the same germ plate.

The impression received at operations is that the dural sac lies freely in the bony vertebral canal, communicating with the wall of this canal only through the roots and vessels. Ventrally, the dura mater is loosely attached to the posterior longitudinal spinal ligament (Lewin, p. 606).

Very careful anatomical study made by von Lans, later complemented by Felten, has shown that a plexus of innumerable minute fibres stretches between the dura and the arachnoid These strands of tissue or fibres are scarcely visible at operation; but in photographs taken under special lighting arrangements they appear to be demonstrable (Felten) Apparently, the roots are suspended in the same way from the root sheath These strands fan out into the structure of the dural sac, in which small reinforcing ligaments are thus formed, enclosing the site of entry of the roots. Eventually these structures merge in the filum terminale. Were these fibrils to be noticed at operation, it seems to us that they would obviously be taken for adhesions.

However, both at operation and at autopsy, conditions have been observed in which there was pronounced accumulation of tissue outside the dura. Epstein calls this fibrosis or fibrolipomatosis. Signs of inflammation were not always found. Sometimes the degree of fibrosis is such that the roots are constricted. He knew of eighteen cases in the literature in which the patients not only complained of pain in the back, but in the extremities as well.

Fat tissue is encountered, sometimes in comparatively large quantities, but it is not considered to be pathologically significant. Nor is it implied that there is any connection between the quantity of fat present and the available space, so that a wide vertebral canal with a wide or narrow dural sac may contain much or little fat. Obviously, a wide vertebral canal can contain more fat than a narrow one, but this does not mean that a wide vertebral canal containing a small dural sac must necessarily be filled up with fat tissue. The accumulation of fat may, however, become pathological if it takes the form of a lipoma, *e.g.*, *associated with spina bifida, an epidermoid cyst or congenital skin sinus.* In the latter cases the lipoma can be situated both extra- and intradurally. Congenital accumulations of fat are seen in association with spina bifida and especially

with tumours. Boeters writes in his book on *Erbbiologie* (p. 295) that fat tissue may have accumulated in certain places in the body under the influence of endogenous factors. He assumes that the distribution of fat in the body is determined genotypically. This may provide an answer to the question as to why the epidural space sometimes is and sometimes is not filled up with fat, without any such implication as the general nutritional condition of the body or the width or diameter of the vertebral canal.

Both the connective tissue and the fat tissue are liable to become inflamed. Such inflammations may take on any gradation from slight to severe adhesion of the root, mild epiduritis developing into, or forming the residual condition of, an epidural abscess. Inflammation of the tissue is called epiduritis or epimeningitis, sometimes intermeningitis, in the literature, and is apparently by no means rare. Biemond and Prick have described several cases of epimeningitis in the Dutch literature, with abscesses forming in the epidural space. Amzeleg collected 299 cases from the literature which he held to be primary epiduritis. Epstein reviews the matter fully, mentioning several authors. Raudam describes treatment with epidural Procaine infiltrations, to which 40 cases of radiculitis-epiduritis reacted favourably. Nils states that, in conjunction with an inflammation of epidural fat, signs of inflammation were also observed in the roots and in the ganglia. Biemond and Prick studied this phenomenon in animals. By lumbar puncture they introduced micro-organisms into the cerebrospinal fluid and, after a time, saw epimeningitis develop which spread over several intervertebral discs and thus precipitated a hernia nuclei pulposi of infectious aetiology.

Finally, epimeningitis can also result from disorders resembling inflammation of the vertebral bodies or discs. The inflammation may be specific, or proceed from a non-specific osteomyelitis. Meanwhile, several cases have been published of post-operative epiduritis, said to have derived from a surgically treated disc. Brussatis observed four cases of it out of 2000 patients operated upon, without finding evidence of lesions in the cauda equina. Lenshoek published three similar cases with radicular pain, in which micro-organisms were detected. He expressed it as his opinion that the same complications might be involved in a simple lumbar puncture and after discography. The latter possibilities are also pointed out elsewhere; but micro-organisms are not invariably found. Turnbull, for instance, points out that creeping epiduritis may develop after a disc operation, from which no micro-organisms can be cultured, while the cerebrospinal fluid undergoes lymphocytic changes similar to those seen in association with meningitis tuberculosa. The undoubted impression received is that, in his opinion, sterile epiduritis can occur and, becoming chronic, brings about changes in the cerebrospinal fluid.

The lesions of the sliding and supporting tissue in the epidural space, referred to above, are as a rule secondary.

We shall not here enter into the clinical aspect of epiduritis (see Lewin, p. 606). It is painful and causes back and radicular pain; if space is congested, this produces a non-specific myelographic picture.

In the majority of the 18 cases of fibrosis in the epidural space reported by Epstein, the patients complained of referred pain which, contrary to radicular pain, radiated upwards from the calves or thighs. This is reminiscent of the symptoms associated with intermittent claudication and of the ascending cramps in the calf known to be associated with other vascular disorders. Numerous patients not presenting the typical symptoms of dysbasia angiosclerotica do complain of cramp in the calves after exertion; and somehow or other the symptoms are associated with the position of the leg, say raised with the patient supine, or are exacerbated by warmth. This kind of pain is usually associated with vascular disorders and should therefore be distinguished from typical radicular neurogenic pain. We now begin to suspect that the blood vessels in the epidural space are also affected in the cases of fibrositis reported by Epstein.

In the cases of epimeningitis described by Biemond and Prick the patients likewise complained of cramp in the legs; both authors surmise that this is of vascular genesis.

The syndrome resulting from vascular disturbances in the epidural space has been studied by Déjérine (*Intermittent Claudication of the Spinal Cord*) and Ramsay Hunt (*Arteriopathic Disease of the Lumbar Arteries or Abdominal Aorta*) (See Kinnier Wilson, p. 1174).

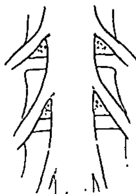
Whereas epiduritis and pathological accumulation of fat are predominantly situated dorsally, lesions of the vascular system will commonly be found on the ventral side of the dural sac. *The vessels in the epidural space* consist of a few arteries and many veins.



The *arteries*, together with the roots, as side branches of the spinal artery, enter the vertebral canal through the foramina. About three of the four roots are provided with one of these accompanying arteries. (Felton and von Lans). This bears out our own experience during rhizotomies in the root sheath; in about 3 of 4 cases we saw an artery running over the radices within the root sheath. This little artery accompanying the internal radix in the root sheath penetrates into the root sheath at approximately ½ cm from the place where the root enters the dural sac.

In addition to the arteries accompanying the roots, there are also elongated arterial vessels to be seen running lengthwise with the dural sac. They are situated very much laterally against the wall of the vertebral canal. They anastomose with the arteries entering over the roots from the foramen and also with the arterial blood vessels on the opposite side. They do not always occur; or at any rate are not observed in every case. It is our impression that a root which does not receive its own arterial blood vessel from the foramen is supplied by these elongated anastomosing arteries. If our observation is correct, these arteries often cross the L.5 root close to the entrance to its intervertebral foramen. Together with accompanying veins, these arteries run like a small band over that part of this root.

Unlike this arterial system, the *veins* of the epidural space form a plexus. This may be composed of blood vessels, varying in number from case to case and also in thickness. The network forms a junction between the venous emissaries at the back of the vertebral bodies and the veins running in a lateral direction via the intervertebral canal towards the big veins outside the vertebral canal. The epidural plexus of veins is thus connected with the pelvic, gastric and thoracic veins and with the intercranial sinuses (Prick, 1955). These emissaries are situated for the main part in the lower part of the posterior surfaces of the vertebral bodies, *i.e.*, just above the lower vertebral margins. They are usually grouped in twos or threes in the centre, between the intervertebral foramen and the lateral boundary of the dural sac. Armstrong describes the venous plexus as consisting bilaterally of two main venous strands with many transverse anastomoses. The veins form a small separate plexus in the intervertebral foramen, thereby sealing off the foramen on the outside (Felten). Batson (1957) describes the epidural venous plexus in detail.



When a venous plexus of this kind assumes abnormal proportions, it is called *varicosis*, or pseudo-varicosis, in the literature. There is no doubt at all as to its occurrence (Cossa, Goinard, Prick, Schmorl, Veraguth, Zukschwerdt and many others); but opinions differ as to its cause, which is not surprising, since it can probably be activated by a variety of mechanisms.

It is necessary, therefore, to distinguish one form of varicosis from another and, first of all, there is *primary varicosis* as described by Ostertag and Schaltenbrand. Both assume, on the basis of their investigations and study of the literature, that primary varicosis results from a disontogenetic disturbance of the mesoderm. Through a disturbance in the development of the spinal cord,

the venous vessels become over-developed per segment (or among several segments). Some authors, including Biemond (1941), subscribe to this. Zukschwerdt endorses this opinion in pointing out that naevi and skin haemangiomas are found in the same segments. Other authors, including Epstein, however, have not been able to observe this coincidence significantly.

Solitary primary varicosis should be subdivided into a local disorder of the vertebral canal: *varices in the epidural space*, and the *varicosis limited to one root* (Mixter, Prick). These varices around one root are said to be congenital; they seem to us difficult at first sight to distinguish from the plethoric venous network around an inflamed radix, or from secondary hyperaemia, say as the result of blood pressure in the blood vessels. Differential diagnosis might be facilitated by the fact that the primarily pathological blood vessel (thin muscle tunic under the microscope) does not readily shrivel upon coagulation; the wall of the vessel bursts open because a hole is burned into it.

Persons of distinctly *asthenic habit* have an increased tendency towards sanguinolence when undergoing manipulations in the epidural space. This, in our opinion, is due to the quality of the vascular wall of a too luxuriant venous plexus.

Secondary varicosis is said to be precipitated by hyperaemia or blood pressure in the blood vessels. This hyperaemia may be the result of

(*a*) a *local* lesion in the vertebral canal itself; for, if the avascular disc tears, tissue proliferates in it, according to Harris and MacNab and Schmorl, and vessels secondarily invade the disc.

(*b*) *Inflammation* within the spinal canal is also supposed to lead to hyperaemia (Krischek, Zukschwerdt).

(*c*) Plethora of the plexus may result from hyperaemia *outside the spinal canal*, such as following upon tumors and inflammation in the abdomen and gynaecological disorders.

(*d*) Varicosis may be the result of over-abundant blood supply, *e.g.*, through *vascular tumours* (Suter), angiomatosis of the central nervous system (Prick and Woltering) and haemangiomatosis vertebrae (Epstein, Prick). That this latter possibility is not imaginary becomes clear on learning what Topfer (quoted by Epstein) has to say. At autopsy he found haemangiomatous lesions in the vertebral bodies in 12 % of 200 cases. This statement is, admittedly, received with reserve by other authors, on the supposition that Topfer's findings often related to very minor disorders, while, moreover, it is necessary to bear in mind that some of these angiomatous disorders might possibly be aneurysmatically distended bone cysts Yet it would seem to appear from the survey found in Epstein and elsewhere that these disorders occur far more often than they are diagnosed primarily by X-ray or at operation.

Hence haemangioma vertebrae brings on symptoms, not only on account of its tumour-like growth (erosion of the bone and thickening of the pedicles of the arch → root compression in the foramen: Brocher, p. 49), but also by congestion of space as the result of secondary varicosis. Moreover, angiomatous symptoms of a general nature may occur, due to disorganised vascularisation of the roots.

Plethora of the epidural plexus through blood pressure in the vessels is another possibility. This may result from a *general disturbance of the circulation*, as in failing compensation. Cases in which patients complain of indeterminate pain in the back and are found, upon examination, to be suffering from heart disease are by no means few. In those sporadic cases in which myelography was resorted to on account of the intensity of the symptoms, non-typical recesses were found for a hernia nuclei pulposi.

With the patient prone, in an exaggerated kyphotic posture on the tipped operating table, venous hyperaemia can be induced by *congestion in the vena cava*: it abates or disappears when the abnormal posture is rectified and the table is flat again.

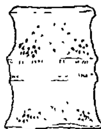
Increased blood pressure in the lumbar venous plexus could quite conceivably originate from a *disease of the chest, e.g.*, carcinoma of a bronchus. Cases are known of patients complaining of indefinable, vague back trouble who were later found to have a tumour of the lung. Lastly, increased pressure may be the result of a *local obstructing change in the vertebral canal* itself. We are thinking primarily of a hernia nuclei pulposi and pronounced lipping, one result of which being that the venous emissaries are constricted and occluded. Helander and Lindboom (Spiegel 1957, p. 309), using a technique of femoral vein catherization with abdominal compression, report obstruction of the internal vertebral venous plexus and veins in the intervertebral foramina and prolonged filling of the pelvic veins (in a number of lumbar disc herniations). Although the assumption of this local cause of a plethoric venous plexus is repeatedly found in the literature, our comment is that it is difficult to imagine so extensive a network of anastomoses being obstructed locally in this way. On the other hand, it is a fact that venous filling ceases the moment a large herniated disc or pronounced lipping has been removed. According to Zukschwerdt, it was observed at operations that a non-pulsating dural sac immediately became active again after the elimination of a large impediment, like a herniated disc. Considering that, as corroborated by numerous investigations, minor abnormalities are found in the cerebrospinal fluid in association with hernia nuclei pulposi, which might be the result of occlusion; also considering that it is hard to account otherwise for these minor increases in protein and multi-

plication of cells, we have nevertheless to ask ourselves whether a local impediment like a herniated disc, etc., may not, after all, obstruct the dural sac to such an extent as to precipitate these phenomena over a wide area. After removal of the protrusion, pressure disappears from the occluded part of the dural sac and at the same time the cause of the heightened pressure of the venous system is eliminated. A local obstruction could effect a more extensive affection of the veins by this mechanism.

Plethora of the epidural venous plexus is liable to lead to profuse haemorrhages at laminectomy. A strong tendency to sanguinolence is also associated with *osteoporosis of the vertebrae*. In any event, the amuscular vessels in bone tissue remain open: the haemorrhages from the osteoporotic bone are more intense and are not readily stopped by plugging. It is also our impression that in osteoporosis the veins of the epidural plexus are wider than usual.

The clinical picture of varicosis of the lumbar epidural space is not specific. It should be suspected in corpulent individuals, mostly women with slightly high blood pressure and ectatic veins in the legs. Usually the neurological picture is poor in symptoms. There is no pronounced fixation of the vertebral column, while the complaints of low back pain referred to the legs are described in vague and inconsistent terms.

Causalgia is said to be induced by irritation of nerve fibres in the vessels. (?) Symptoms simulating dysbasia derive from derangements of the vasa nervorum. Confronted with patients presenting these symptoms, the practitioner should have the possibilities mentioned above in mind. In radiographs the number of emissaries is sometimes seen to have increased, to such an extent, indeed, on occasion that the structure has been described as "pile-worm vertebra" (numerous perforations made in old wood by wood-worm). If myelography is resorted to, little or no importance should be attached to an ill-defined opacity, even if very large, in the contrast medium, if this does not correspond to a typical recess in a posterior direction on the lateral myelograph at one particular level which corroborates the clinical syndrome. If one should proceed to explore in such a case, the likelihood of finding a nuclear retropulsion would be comparatively remote. After the surgical treatment of varicosis (coagulation-extraction), the symptoms may be expected to persist or to return in a very short time, *viz.*, in two to six months, the decompressive effect of the laminectomy passes and the plexus of vessels, if extirpated, grows again. This, in fact, is fortunate, for, with the renewed ingrowth of the vessels, the indeterminate, cramp-like pain in the calves, which may remain as a post-operative



complication of any laminectomy as the result of manipulation of the vessels in the epidural space, also ceases.

The shape of the vertebral canal

The spinal canal might be described as a smooth tube covered on all sides with ligaments.

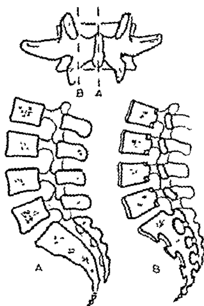
The *anterior wall* is formed by the vertebral bodies and the intervertebral discs, locally covered with periosteum and the posterior longitudinal ligament.

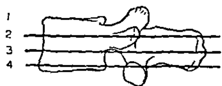
Its *lateral wall* comprises the foramina, the articular processes and the joints, segments of the laminae and pedicles, all these parts being interconnected by ligaments.

The *posterior wall* also consists of ligaments, chiefly the ligamentum flavum, which merges locally into periosteum on the ventral side of the arch, the bony part of the posterior wall consisting of the arch itself and its lamellar extensions.

It will therefore be seen that the laminae form part of the lateral as well as of the posterior wall, which is due to the fact that they cover the vertebral canal in a concave curve.

A single cross-section does not suffice for an impression of the boundaries of the spinal canal; for, in *sagittal* cross-section through the mid-line it looks as though the posterior wall were formed by arches connected with the ligamentum flavum. But in *parasagittal* section, *e.g.*, where the roots run at the entrance to the intervertebral foramen, one finds, in addition to the arches and ligamentum flavum, extensions of the lamina and of the articular processes. At the site of the entrance to the intervertebral foramen we called this space the "approach to the intervertebral foramen" (= lateral recess) In the vertebral foramen itself this space is called the "sulcus lateralis" in the literature. It is likewise clear that in a *transverse* section there are different boundaries, depending on where this transverse section is made. If it is exactly through the arch, the resulting cavity is described as quinquangular, the five angles being formed by the posterior aspect of the vertebral body, the two pedicles and the two lateral parts of the vertebral arch standing at an angle opposite to each other. If this transverse section is made below the arch,





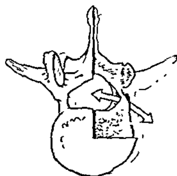








bone tissue will no longer appear in the median line; the shape is then erratic and, roughly speaking, it has become triangular. The base of this triangle is formed by the posterior part of the vertebral body; the sides, on the contrary, are open at the site of the intervertebral foramen, vaulted as they are by parts of the articular processes and the laminae; dorsally there is nothing except the ligamentum flavum. Taking the section a little lower still, *e.g.* exactly where the ligamentum flavum stretches from foramen to foramen, the only boundaries will evidently be the back of the vertebral body and the two openings into the intervertebral canal, the whole being dorsally covered by the ligamentum flavum. Lastly, a transverse section at the level of the disc (hence generally immediately above the arch) will show boundaries formed by the intervertebral disc, extensions of the pedicles, which merge into the superior articular process, and then dorsally again only the ligamentum flavum.



With these cross-sections before us, the course of the *sulcus lateralis*, where it merges into the approach to the foramen, becomes clearer: in the vertebral foramen the sulcus is bounded laterally by the pedicle and dorsally by part of the arch. Caudal to the vertebral foramen the sulcus is roofed by the lamellar extension of the arch where it merges into the inferior articular process. The lamellar extension of the top of an arch in the superior articular process lies ventrally to the lamellar extension of the base of the arch (situated at a higher level) in the inferior articular process and is therefore closer to the root. The most ventral lamella, which we sometimes call the "pantile extension of the pedicle", roofs the approach to the foramen immediately in front of the foramen, lies closest to the root and is, therefore, best equipped to modify its course. Hence the shape of the vertebral canal cannot legitimately be said to be determined by the shape of the arch only. It is precisely at the site of the foramen that the positions of the joints, the lamellar extension of the arches and the pantile extension of the pedicles of the arches will govern the shaping of the canal.

Perhaps it is for this reason that the shape of the lumbar vertebral canal is represented so variously in the literature. Krayenbühl, Schmorl and Spalteholz describe it as being oblate cervically, round thoracically and triangular in the lumbar region. For the lumbar region Armstrong describes variations from oval to triangular and trefoil. Verbiest points out that the greatest extension of the arch lamellae is below the level of the arch itself, so that the shape much depends on them at this place, as has been observed in myelograms. Apart from this, it should be borne in mind that the diameters of the vertebral canal in the foramen of the five lumbar vertebrae differ considerably one from the other. Krayenbühl and Zukschwerdt refer to Dubs' detailed descriptions; and he stressed the fact that the space at L.V–S.I is entirely different from that at the lumbar vertebrae situated at a higher level. Schmorl mentions Fineschi's findings. The latter shows how very different is the shape of the spinal canal at L.V–S.I in the case of transitional vertebrae.

The literature gives exact measurements of the diameter in the antero-posterior direction and between the pedicles. Huizinga, Thomson, Van der Heiden and Vinken took these measurements from skeletons. Verbiest took measurements during operations, while Elsberg and Dyke measured the interpedicular distances on radiographs. From these it transpired that the spinal canal is narrowest at the level of L.III and L.IV and its width is somewhat greater than its depth. Slight antero-posterior increased size was found at the level of the L.V vertebral body in the transition to the S.I vertebral body, but an enormous increase in the width of the vertebral canal. It strikes one as extraordinary that the antero-posterior diameter of the L.I and L.II vertebral bodies should be greater than the width there (Huizinga). It may possibly be because the transition here from the round thoracic to the triangular lumbar shape is only gradual; nor should it be forgotten that normally the spinal cord reaches to this level.

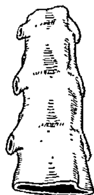
At L.IVand L.V the vertebral canal appears gradually to assume a sacral



shape caudally. The round thoracic has already become the trefoil and triangle, which, further down still, are transformed into the flattened slit similar to that in the sacrum. This latter is possibly due to the fact that the lower roots, like S.1, S.2, etc., take a more perpendicular course beside the dural sac in a sagittal direction.

The higher lumbar roots run more obliquely and then disappear out of the vertebral canal. Owing to their perpendicular sagittal course, the lower (lumbar) roots require room in the vertebral canal themselves, whereas the theca, after the big roots have left it, no longer needs the ventro-dorsal size it required at a somewhat higher level.

We have observed that the vertebral canal at L.IV–L.V, but more especially at L.V–S.I, may already have assumed the flattened configuration which is appropriate to the deeper sacrum. Fineschi and Schmorl, as we have seen, described this in association with transitional vertebrae, when it is, in fact, strikingly apparent. But, without any other manifestations of a transitional vertebra, this exaggerated oblate form of the vertebral canal can be seen at L.V–S.I; we consider this to be in itself a sign of sacralisation. One might call it a *partical sacralisation*, evident only by the shape of the vertebral canal.



The lumbar spinal canal cannot be compared to a rigid flat tube, but rather to a lead pipe with many indentations (lamellae, joints, disc and so forth) roughly modelled to a triangular shape, with the outlet pinched in. This is, more or less, the shape of a "normal" vertebral canal, but its irregularity is accentuated when pathological conditions prevail. Lindblom draws attention to the loss of room and consequent change in shape of the bony vertebral column attendant upon arthritis, arthrosis and spondylosis. Bradford and Spurling, Lewin, Love and Spurling described the protrusion of the joint facets into the lumen of the canal and call it the "facet syndrome". Chapchal, Hantaets, Serpijener and Verbiest referred to lamellar extensions and pantile extensions of the arch and pedicles. Although these three things are not identical, they are interrelated. In association with arthrosis a facet will protrude further into the canal and will also do so if predisposed by the extension of the arch and pedicles. Descriptions of posterior lipping, chondrosis and nuclear herniation, as so many factors liable to affect the shape of the vertebral canal, are legion in the literature. To these could be added: the contorted arch due to a disease of the bone or an injury, haematoma, oedema and inflammation. Each in itself, or in conjunction, leads to limitation of the physiological space in the spinal canal, they might be compared to blows of the hammer on the lead tube. It depends on the intensity and number (age), but not less upon the place where these hammer blows fall, whether the contents (roots) are upset by them.

When available space is entirely filled by a pathological process (tumour, inflammation, residual condition of a trauma or large hernia), we speak of an

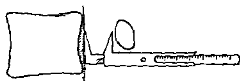
absolutely stenosing process. Obviously, a mechanical irritation can proceed from this. But, as the deeper knowledge acquired of the pathology of the vertebral column raised doubts as to whether such a variety of complaints could be caused in so simple a manner by a mechanistic factor, the idea of *relative stenosis* began to take hold. We mention the following authors who have written on this matter: Bradford and Spurling, Chapchal, Cossa, Fineschi, Hanraets, Haverkamp, Krischek, Larmon, Lewin, Love, Magnuson, Schmorl, Thurel, Verbiest, Zukschwerdt.

Although very different views are held as to how the various factors may produce relative stenosis, what is meant by this mechanism is clear enough: a certain abnormal shape of, or change in, the vertebral canal may restrict the available space to some extent, without, however, necessarily producing symptoms, as some spare room is left. An incidental circumstance may, in turn, cause some congestion and fill up the available spare room. The two factors together may produce absolute stenosis with resulting symptoms. (*E.g.*, a small tumour in a narrow vertebral canal, a small herniation or lipping, if situated in an otherwise physiological intervertebral canal, a small hernia nuclei pulposi plus lipping plus oedema plus an arthrotic joint in a narrowed canal, etc.) The first factor present then predisposes to absolute stenosis; it may be endogenously determined or acquired. We can now see how important the endogenous morphogenesis of the spinal canal may be to the tracking down of endogenously determined causes of low back pain and radicular pain. This, after all, is what we set out to do.

The dimensions generally

In the ensuing pages we shall constantly be referring to the width or the narrowing of the bony vertebral canal. The need was felt for some standard yardstick, against which any discrepancies could be compared in exact terms, for the difficulty is to know what may legitimately be called too wide or too narrow. A standard is obtained by taking exact measurements in a large number of *normal* individuals, thus obtaining a mean with extreme values for the smallest and the largest size. This leaves us with another question on our hands, *viz.*, how is "normal" to be defined? When Elsberg and Dyke attempted to establish a standard measurement in two hundred skeletons, stenosis of the vertebral canal as a clinical entity was not known, nor were the special aspects of a spinal canal branded as too wide. Thus Huizinga was able to establish the minimum size – in the antero-posterior direction as well as otherwise – of the vertebral foramen after measuring 51 skeletons, finding 19 mm. (If, by chance, Huizinga's material had included the skeleton of one of Verbiest's seven

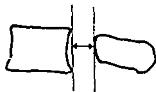
patients in whom a 1-mm smaller antero-posterior diameter was found in one measurement of one vertebra than Huizinga's lowest figure, it would have meant that Huizinga's minimum value would have had to be lower by 1 mm.) Understandably, therefore, both investigators come to the conclusion that the idea "too narrow" or "too small" is to be taken in a relative sense and that there are no watertight grounds for speaking of an absolute narrowing. We have to consider the standard dimensions resulting from the work done by Elsberg and co-workers and Huizinga and co-workers as obtained from unselected material. They established this standard dimension by assuming that all their specimens were "normal", but we may with as good right suppose that this was a combined collection of normal skeletons and variations not recognised as "abnormal". A correct standard could be obtained if the extreme cases could be selected from such material. One would start at random by eliminating the largest and the smallest sizes. A selection would be to some purpose only if the material selects itself, as it were, in that many cases with extreme values present a common syndrome. Now it would seem that Verbiest's material meets the case, for he noted that a certain syndrome was associated with *persons having extremely small antero-posterior diameters* of the vertebral foramina. If these, as found at operation, were significantly smaller than the extreme values found, say, by Huizinga in a larger group of skeletons which he considered normal, one might be justified in assuming that Huizinga's dimensions represented the normal and that the dimensions found by Verbiest in his patients can be said to be pathological. However, upon comparison this was found not to be so, as only two patients in Verbiest's thus selected group were one mm below Huizinga's minimum value in one dimension of one vertebra. This beats out the view of both these investigators that one cannot at present speak of too narrow, or too small, or too large, in an absolute sense.



As this matter carries great weight in the possible establishment of endogenously determined stenoses, it is to the point to consider the *technique by which the measurements were taken.* Verbiest uses an instrument, specially made for the purpose, consisting of two feet, which is placed during operations between the arch and the posterior surface of the vertebral body. In this way he measures the antero-posterior diameter of the vertebral foramen, the ligaments having been previously prepared on the ventral side of the arch. The deeper limb of the instrument is placed on the ventral side of the dural sac, hence between the dural sac and the longitudinal ligament. Thus the

physiological concavity at the back of the vertebral body is not neglected, but neither the thickness of the periosteum, the posterior longitudinal ligament nor the venous plexus there is included in the calculation. For this reason, ¾ mm is added to the size measured; however, this is an unreliable correction. Although the plexus of veins and the periosteum are admittedly already very thin, the ligament may vary considerably in thickness. This is further accentuated by the fact that, precisely in the median line, the ligament is apt to run in thick bands. Paying special attention to this, we have measured (after extirpation) variations between ½ mm and 3½ mm. If the ventral limb of the instrument were placed on or beside a thick bundle of, say, approximately 3 mm, discrepancies of about 3 mm would be obtained. As the dimensions with which we are concerned are of the order of 11, 12 and 14 mm, such variations would affect the measured diameter to the extent of roughly 25 %. The dissecting out of the ligaments on the ventral side of the arch and the addition of ¾ mm as a correction for the thickness of the longitudinal ligament make this technique in actual practice a means of measuring from bone to bone. Let the thickness of the longitudinal ligament be 2¾ mm, then, according to Verbiest's method, the bony diameter of the vertebral canal in the antero-posterior direction is short-measured by 2¾−¾ = 2 mm. On the other hand, ¾ mm is unnecessarily added to the measurement between the arch and the longitudinal ligament (not counting the almost negligible thickness of the periosteum at the arch). Thus the membranous diameter is too large by ¾ mm and it is precisely the distance between the soft parts (the functional diameter) that we want to know. After all, the lining, it thick, of the vertebral canal likewise plays some part in limiting the available space.

Huizinga and co-workers took measurements from skeletons, a correction being made for the physiological concavity of the posterior surface of the vertebral body (which is neglected here). They described and illustrated the distance measured between the ventral side of the arch and the connecting line



between the superior and inferior vertebral margins. This means that the connecting line runs over the concave posterior of the vertebral body, at a distance of several mm from the vertebral body itself. Thus the distance between the arch and vertebral body, or bony vertebral canal, is not exact, *i.e.*, the recorded measurement is short. Hence neither technique is exact. The results of the two methods are comparable in the sense that both approximate the size of the bony vertebral canal and that circumstances inimical to accuracy (thick ligament and deep concavity of the vertebral body) produce

errors of the same kind (sizes short of the true dimensions) in both methods.

We would say that an unusually deep concavity of the vertebral body is more common than an unusually thick ligament. Perhaps this might account for the fact that the dimensions of the foramina found by Verbiest to be narrowed are within the "normal" (in our opinion, somewhat too low) values measured by Huizinga.

Interpedicular distances in mm

	Elsberg and Dyke			Huizinga and co-workers [1,2]		
	"Normal"	Extreme values		Mean	Minimum	Maximum
L.I	23-28	20	33	23.4	19	28
L II	24-29	22	33	23.4	20	26
L III	25-30	22	35	23 5	20	28
L.IV	25-31	23	35	23.5	19	29
L.V	28-33	24	39	23.8	20	32

[1] Huizinga, 1951
[2] Verbiest, 1956

Comparing the distances between the pedicles of the arch found by Huizinga with those measured by Elsberg, we see that the latter's are on the whole larger. This cannot be due only to the fact that Elsberg's X-ray material comprised more specimens than Huizinga's number of skeletons and therefore provided more scope for extreme values. Moreover, Elsberg's minimum values are also higher than Huizinga's. The reason for this difference is that Huizinga took his measurements from skeletons, omitting the concave posterior of the vertebra, whereas Elsberg performed his on radiographs. No doubt the inevitable projection error in X-ray exposures is also responsible for this incongruity. Calculations have shown that a radiograph for which the instrument was placed at 6 m is already 4 % out of drawing. We do not know by how much more the image is out of drawing when the instrument stands at the usual distance of about 0.8 m or 1.20 m, but the percentage is likely to be appreciably higher

As they stand, the results obtained by these two techniques cannot be made to agree. Measurements made on radiographs have only a relative value and can only be used to good effect for comparing certain dimensions under identical circumstances

For all that, the results of these three methods are valuable, inasmuch as they give us some idea of the conditions within the vertebral canal. But, in

an absolute sense, they are unreliable and do not tell us whether a size is absolutely too small or absolutely too large.

In view of the unreliability and relative value of the existing methods, we did not take measurements during our operations; nor did we feel competent to devise a new and more exact method to take their place.

Variations in the dimensions of the vertebral canal may lead to:
a narrow vertebral canal;
a wide vertebral canal, or
an asymmetrical vertebral canal.

Stenosis of the vertebral canal

Sarpijener was the first to describe narrowing of the vertebral canal (in 1945 and 1947). Especially in young children he found congenital stenoses, both isolated and in combination with spina bifida, sometimes of such severity that insufficient room was left for the spinal cord, which was then reduced to a thin thread. Sarpijener's stenosis is the only form of absolute stenosis. He describes annular structures around the spinal cord at one or several levels and extensive narrowing of a major part of the spinal canal. These lesions might be associated with enuresis and deformity of the lower extremities, as well as with spastic paresis. We shall not further discuss these extreme cases here, since they are exceptional.

It is much to Verbiest's credit that he should have noted that an incongruity between the capacity (product of the dimensions) and the contents of the spinal canal may give rise to a circumscript syndrome in adults. As far as we know, he was the first to define so clearly in the Dutch literature, and probably in the world's, the relation between endogenously determined – subclinical – changes to an apparently normal spinal canal and the symptoms they produce. As the result of the recognition of this relationship and the definition of this syndrome, several patients in the past have been relieved of their disabilities by appropriate therapy.

It is immaterial, as far as the theory is concerned, that the syndrome defined by Verbiest is confined to exceptional cases. It is also by the way (though, to our mind, of added value in helping us to a better insight into the matter) that the author had to modify his initial view, in that later on exact measurements had shown that the dimensions of only a few of his stenosed canals were below the minimum which had since been established for the "normal" vertebral canal. In other cases, with a narrow, but not abnormally narrow spinal canal, he assumed that symptoms of compression were caused in the ultimate by incidental conditions (small disc sill or posterior lipping). He thus amplified

the range of the syndrome he had detected by making it also dependent upon a relative – predisposing – stenosis, thus reverting to the terminology of his first publication on the subject (1949): *La sténose osseuse relative*. Verbiest describes (from 1949 to 1955) first seven cases during the operation of which he received *the impression* that the spinal canal was too narrow. In a subsequent series he mentions two cases of smaller antero-posterior dimensions of the vertebral canal than "normal" found by exact measurement during operation. In the four remaining cases he discovered, again by exact measurement, that, although these same dimensions were exceedingly small, they were just within the physiologically normal minimum of the control material (Huizinga). He quotes two cases from the literature reported by Van Gelderen which were treated with the same success in the same way for the same symptoms as his patients, but without suspected anomalous dimensions of the vertebral canal.

Finally, we find a reference in Spiegel (1955, p. 339) to Scott, who had treated similar cases surgically and had come to the same conclusion. Scott surmised, however, that a moderate median prolapse of the disc plus thickening of the ligamentum flavum might help to produce symptoms of compression in an already narrowed canal. In the first seven and subsequent two cases in which Verbiest assumed a true stenosis of the vertebral canal, he did not observe any adventitious precipitants, such as hernia, posterior lipping, changes in the joint or a distended ligamentum flavum. In four cases of the last series he describes a (relatively) narrow canal, to the narrowing of which a small hernia, posterior lipping or protruding joint facets contributed. All these patients had this in common that they had suffered for many years from some form of low back pain, radicular pain varying in intensity and often bilateral sensory disturbances or paraesthesia. These symptoms were brought on by strenuous exertion, like prolonged walking or standing, and *ceased instantaneously when the patients rested*. They complained further, under the same circumstances, of fatigue and loss of power in both legs; after walking some distance, they had to stand still on account of severe cramp in the calves, but were able to proceed after a short rest. The nature of the symptoms suggested vascular disorganisation and the patient was suspected of, and treated for, intermittent claudication All the patients were men of middle age who had never experienced anything of the kind in their youth. Radicular pain persisted during rest in some patients of the last series, but in those cases an incidental root lesion was found and there were also symptoms of neural loss before the operation. On the whole, however, the neurological picture was poor in symptoms and *not* identical with the classical syndrome of hernia nuclei pulposi There were minor disturbances in sensibility or abnormal reflexes, very occasionally slight paresis or atrophy.

but as a rule the Lasègue test was not clearly positive. There was thus an incongruity between the paucity of symptoms in the neurological picture and the severity of the persisting symptoms. After myelography, a constant stop or very slow passage of the contrast medium was found somewhere in the lumbar vertebral canal on the radiographs in all cases. The clear, colourless cerebrospinal fluid contained few cells and a slightly to moderately increased percentage of protein. As a rule, the mobility of the vertebral column was limited to some extent in all directions. It is reported that, although pressure precipitants, like coughing, sneezing and straining, occasionally exacerbated the symptoms, this was more often only so in cases where other space-occupying processes as well were found at operation.

All these cases were treated by decompressive laminectomy and, depending on what was found, facetectomy, extirpation of a hernia or chiselling off bony edges. All these patients, who, where possible, were followed up for many years, were relieved permanently of their symptoms after the operation. Anomalies, taking the form of spina bifida and lysis of the arch, were discovered in the spinal column. On several occasions the dural sac was found to be abnormally narrow, even in proportion to the already narrow spinal canal. In those cases the dura had to remain open after inspection. Before removal of the arches, the dural sac showed no fluid pulsations. As the dural sac contained roots only and lacked fluctuating fluid, it was like dough to the touch. After removal of the arches, in most cases the dural sac began to unfold spontaneously. Another anomaly observed was a cystic distension in the form of an epidural cyst. Transverse measurements usually showed normal distances between the pedicles, though sometimes very considerable interpedicular distance and very occasionally an abnormally large size (on the radiographs).

The evidence of anthropometric examination (Verbiest, 1951, pp. 1968 and 1970) is that stenosis is not a manifestation of a general disturbance in the growth of the skeleton, but must result from a local process of which the cause is not yet known. In 1956 (p. 1610) Verbiest presumed that stenosis of the vertebral canal was probably congenital. To put it at its mildest, one might say that an impediment to growth, probably determined endogenously, became operative before adulthood.

No signs of skeletal diseases were found at histological examination. Two of the patients were brothers, which might point to a "developmental disturbance" (Verbiest, 1956, p. 1610).

Verbiest refuted the suggestion that the myelographic abnormalities might be caused by arachnoiditis when he did not find this upon intradural inspection.

It is not yet clear why the symptoms should be presented *only on standing and*

walking. Possibly a valve mechanism is responsible, through which fluid accumulates distal to the narrowed region when the subject stands and walks. Verbiest noticed a mechanism of the kind during the operation when the patient made straining movements. This explanation, however, seemed still to be entirely hypothetical.

The following comes from our personal experience: A patient, operated upon for a stenosis syndrome, was able to simulate the pre-operative symptoms by straining during the operation

During the injection of 5c.c. of Novacaine into the dural sac, before the stenosis had been removed, the same symptoms were presented; after the decompression, the phenomenon could no longer be repeated through the effect of the Novocaine.

Patients without stenosis experience no sensations from the introduction of 5 c c of fluid into the lower section of the lumbar sac.

We imitated the above experiment in 5 patients who did not present the syndrome of stenosis by carefully plugging their vertebral canals with cotton wool under the L.IV arch and then introducing 5 c c. of saline solution intralumbarly under light pressure. Twice the patients showed signs of distress similar to those attendant upon the stenosis syndrome. (Fluid had not been drawn off first; the L.V arch and the ligamentum flavum were still intact, except immediately under the L.IV arch.)

This experiment may possibly suggest a means, after further experience has been gained, of establishing the diagnosis of spinal canal stenosis pre-operatively by intralumbar injection of saline solution.

It is difficult to explain how symptoms can be produced which are so deceptively similar to those associated with intermittent claudication upon occlusion of arteries of the lower extremities. We have sometimes heard this syndrome referred to as the "syndrome d'effort". We wish to point out that, if this should be confused with Lewis's "effort syndrome", associated with neurocirculatory asthenia (Ned. Tijdschr. v. Geneesk., 1955, p. 871), it might be misleading

Verbiest (1950) discusses this pseudo-intermittent claudication in detail. He reports intermittent claudication of the spinal cord, as described by Déjérine and Sottas They found no changes in the peripheral arteries, but believed it was a symptom of obstruction of the afferent vessels of the spinal cord itself. Hunt described a "lumbar type of intermittent claudication" and suggested sclerosis of the lumbar arteries as the cause of these symptoms; but he could not adduce much evidence in support of this suggestion. This is an inducement to repeat that Jelsma and Ploetner (Epstein, p. 102) found 18 cases of fibrosis around the dural sac and roots, the patients experiencing pain radiating from the calf to the hip. It has been postulated before that this fibrosis constricts blood vessels in the epidural spaces Possibly Hunt implied the same thing by his sclerosis of the lumbar arteries as the authors just mentioned by epidural

fibrosis. Another possibility is that one is the consequence of the other.

Epstein (p. 417) demonstrated the relation between a narrow vertebral canal (at L.IV and L.V) and epidural adhesions. After clearing these, he found that the dural sac began to pulsate again. Finally, we should point out that Duus reported severe injury of the roots as the result of endarteritic processes and thrombosis in the vessels around the roots. These vascular changes were brought about by mechanical pressure (Zukschwerdt, p. 87). Verbiest, himself, suspected direct pressure upon the vascular system, or vascular spasm due to irritation of the sympathetic nerves, as a result of which circulatory disturbances may take place in the fibres of the cauda, leading to loss of function. In his opinion, remission of the symptoms when the patient is supine is comprehensible in the light of this hypothesis, because in this position the circulation would revert to normal.

At the end of his biometric study of the diameter of the lumbar spinal canal Huizinga writes (1951): "The question remains if the 'primary stenosis' of Verbiest is a stenosis in a true sense for the neurosurgeon".

In our opinion, a disease can be claimed to be neurosurgical if it can be combated by neurosurgical treatment, either alone or with other therapeutic aids. Verbiest vindicated that claim. Huizinga goes further by stating that the antero-posterior size of the vertebral foramen is generally smallest at L.III and L.IV. He thinks it is possible that the antero-posterior dimensions of the foramina in Verbiest's patients were smaller still. Huizinga concludes thus: "It seems possible, however, that the cause of the described discordance between 'capacity' and 'content' of the lumbar vertebral canal must *not* be attributed primarily to the 'capacity', *i.e.*, to a true, abnormal stenosis".

It seems to us that this problem will remain an open question until exact methods of measurement, producing comparable results, have been devised. In the ultimate, a stenosis is absolute only if the canal is completely atresic, or if the ratio of capacity to contents is equal to 1. As long as there is any room to spare at all (ratio of capacity to contents more than 1), the stenosis is, theoretically at least, relative. That this exists, and may possibly be caused endogenously, is evident from Verbiest's published papers.

Dilatation of the vertebral canal

On encountering a wide vertebral canal, it is necessary to distinguish the distension due to a congesting process from the abnormally large vertebral canal unassociated with other symptoms or demonstrable causes.

The former is quite outside the province of this study. We shall merely mention

that distension of the vertebral canal as the result of a congesting process is usually found to be in the antero-posterior plane, erosion of parts of the arch then sometimes appearing on radiographs. In 1934 Elsberg and Dyke wrote that, in their material, extension of the interpedicular distance only occurred at the site, and as the result of the growth, of neoplasm. In the same year, they, together with Brewer, pointed out that pseudo-tumours, such as epidural cysts, were likewise liable to cause this dilatation, as well as erosion, atrophy, thinning and extension in a dorsal direction of the arch.

Although the distension in the presence of an epidural cyst could also be regarded as the result of a developmental disorganisation, it is in a different category from those dilatations incidentally coinciding with anomalies which themselves do not cause dilatations.

Such are: anomalies of the spinal cord, like diastematomyelia and intrathoracic meningocele (Herren and Edwards, Kessel, quoted by Jefferson) Walker suggested that the dilatation of the spinal canal in these cases might be considered to have taken place during the development of the individual. He pointed out that there was no erosion of the bone in the dilated arch.

We also found dilatation of the bony vertebral canal described in association with spina bifida (Walker, Lewin, p. 54) and the association of this dilatation with other developmental disturbances in the vertebrae, such as short or rudimentary spinous processes (Jefferson, 1955). Nearly all the above cases cited from the literature showed dilatation in the thoracic part of the vertebral canal, except for one localisation at L.II (Jefferson). It was always accompanied by congenital anomalies of the spinal cord or of the vertebrae. The dimensions given varied dorso-ventrally in the main; the authors emphasized an increase in the total capacity of the vertebral canal. In Jefferson's two cases the interpedicular distance was also considerable, but not greater than the "normal".

The foregoing can be summed up in the words of the neuro-radiologist Orley, thus: "Occasionally, an increase of the interpedicular distances of a number of vertebrae in the upper *dorsal* region is the result of a developmental anomaly and not of tumor". (Author's italics.)

In a provisional report, Hanraets (1953) described ten cases of a very wide dural sac, all at the level of the lumbar vertebral column between the L.IV and S.I arches. Even then he pointed out that a wide dural sac of this kind might be situated in a wide bony vertebral canal which was far larger laterally than is normal when the dorso-ventral size is small. In cross-section, therefore, it was very broad and oblate. This configuration was at first observed in conjunction with other anomalies, *e.g.*, spina bifida, but it was afterwards encountered repeatedly without any accompanying signs of disorganised development.

At that time scant attention was paid to this variation, but, on mature consideration, the author came to realise that this so-called variation might be one of the manifestations of a type of constitution with significant implications respecting low back pain and radicular pain.

The absence of a typical syndrome with pathognomonic characteristics deters us from calling this a disease or a clinical picture. But, on account of the typifying transverse dimensions, we feel compelled to mention these disorders when discussing the wide vertebral canal. It may, however, be emphasized at this juncture that this dilatation is of an entirely different order from that hitherto described by Jefferson, Orley, Walker and others. These write of an oval, round deformation with a dorsal bulge of the arch, almost without exception localised in the cervical, thoracic or high lumbar region. By contrast, the dilatation we are referring to occurs only in the space between L.IV and S.I, that supremely important region to any study of low back pain and radicular pain. Further, we are speaking of an oblate vertebral canal, *i.e.* a broad one, antero-posteriorly small, especially in its most lateral dimensions; in other words: a wide, flat vertebral canal (see p. 448).

It may be asked whether a small antero-posterior diameter of this broad-flat vertebral canal suggests that Verbiest's primary stenosis should be integrated with the variation we have in mind, the more so as Verbiest (1956 p. 1610) measured normal, wide and once even excessively wide interpedicular distances in association with this stenosis (!) To this could be countered that the stenosis in question was found almost exclusively at the level of L.III and L.IV, seldom at L.II and L.I and exceptionally at L.V. We did occasionally find the broad-flat vertebral canal at L.IV–L.V, but predominantly at L.V–S.I. Lastly, both stenosis and relative stenosis produce a pathognomonic syndrome of symptoms, which the broad-flat vertebral canal does not.

Whether the two abnormalities are related and whether relative stenosis is also involved in the broad-flat vertebral canal, or else whether Verbiest's stenosis is an extreme form of the anomaly which we regard as a type of constitution, is, to our mind, still a matter of conjecture. Perhaps we should be thinking along the right lines if we asked ourselves whether a transitional tendency in a cranial direction could cause the broad-flat deformation at L V–S.I to present itself at a higher level, say L.III and L.IV. After all, such a tendency is generally accepted in respect of other manifestations in the spine (Kuhne). Just because the lumbar vertebral canal is physiologically at its narrowest there (Huizinga), it is conceivable that, let us say, a relative stenosis of pathological significance (Verbiest) might arise through a broad-flat deformation (Hanraets) at that level.

The asymmetrical vertebral canal .

It will be evident that, as the result of relative torsion of the lumbar vertebrae, the lumen of the vertebral canal may change asymmetrically. We were unable to find any reference in the literature to disproportion between the left and right halves of the vertebral canal due to an endogenously determined shape of the canal. Yet it does exist. To prove our contention we have only to recollect that the shape of the vertebral canal is affected by sacralisation of L.V; indeed, the word itself implies that the lumbar vertebra as a whole has assumed a sacral shape. In 3000 radiographs, Brailsford found *bilateral* sacralisation of L.V in 4.7 % and *unilateral* sacralisation in 3.4 %. This implies, therefore, that the vertebral canal will also have assumed a sacral shape unilaterally – hence asymmetrically – in those cases.

While studying the broad-flat vertebral canal, we were struck by the fact that radiological manifestations which may point to it often occur on one side only. If we may infer a type of constitution predisposing to low back and radicular pain from this circumstance, we shall find that the asymmetrical broad-flat vertebral canal is also significant (see p. 453).

Degeneration (Retrogression) of the Intervertebral Disc as a Sign of General Degeneration

Before we probe the literature for opinions on this matter, we would do well to make clear what is meant by these terms.

Wertheim Salomonson says that "a definition of the word degeneration must, in the nature of things, be incomplete, inexact and incorrect". Van der Spek concludes his extensive enquiry into the concept *degeneration* with a cautionary comment on the use of this word, because it has become emasculated by the many shades of meaning with which it has been invested. (We found many of the following quotations in Van der Spek's work).

Degeneration can be understood as synonymous with retrogression and decadence Both words have a negative tendency in common (falling-off – descending line – deterioration), but, all according to the circumstances, they may denote something different. Letterer coins the word "katabiosis" to indicate the negative and dwindling aspect of the process.

"Decadence" in the *ethnological* sense (Bumke) stands for the downfall of doomed peoples. *Biologically* it implies the deterioration of essential functions and diminished viability (Jordan)

In *psychiatry* the word degeneration is used to describe a hereditary predisposition to nervous or mental diseases (Hoche). It is in that sense that

Carp speaks of degeneration as the result of a disharmonious development. Cox expresses the same thing in the words: "a loss of correlative qualities". By defining degeneration as "an inherited and heritable anomaly within the range of variability of the species", he challenged Morel's postulate – long held in respect – that the concept was informed with an aetiologico-biological meaning. Morel believed that acquired characters could lead to degeneration in the species.

Magnan's definition was: "Degeneration is the pathological condition of an individual who, compared with his parents, has a weaker constitution and less power of resistance. This deterioration is manifested by certain signs". Sommer calls these signs "morphological characteristics pointing to degeneration".

Neurology is primarily concerned with "retrogression", *i.e.* the decay of nerve tissue which leads to histological changes (Waller's degeneration). There are, on the other hand, symptoms of (micro) heredo-degeneration, implying the destruction, on a hereditary basis, of neurogenic systems (Bremer, Curtius, Sillevis Smitt). The signs of degeneration are then considered to be monosymptomatic expressions of a heredo-degenerative disposition.

In *pathology*, degeneration is usually taken to mean gradual deterioration (Tendeloo). Roessle defines degeneration in a general pathological sense as "a progressive injury to and diminution of specific characters of the organism, accompanied by impaired ability to develop, adapt and recover".

Using the above definitions, we could in this Chapter speak of degeneration and tissue regression in respect of both neuro-psychiatric and pathological problems. *It might be as well to use the word "tissue-degeneration" in the sense of deterioration or destruction of tissue, and "degeneration" for the neuro-psychic sphere with its associated problems of heredity, disposition and so forth.*

Intervertebral discs occupy 12 centimetres of a vertebral column of 70 cm (Junghanns), which therefore consists to an appreciable extent (17 %) of disc tissue. We know of tumours and inflammation as affecting the intervertebral disc, but also that it is liable to degenerate and thus precipitate nuclear retropulsion.

It is by no means our intention to deal at length with hernia nuclei pulposi in all its aspects as a clinical case, either in this Chapter or in any other part of this study. As far as the Dutch literature is concerned, it will suffice to refer to Ernsting's monograph and the papers published by Biemond, Kemp and others, from among the many authors outside the Netherlands we may mention Armstrong, Bradford and Spurling, Krayenbuhl, Kuhlendahl, Lewin,

Spurling (1958), Waris. On the whole, a hernia is considered to be a mechanical lesion in these publications which, as far as congestion of space is concerned, can be compared to a benign tumour. Our task is to discover to what extent degeneration of disc tissue takes place, to what extent this is the cause of herniation and whether this degeneration of the tissue can be taken as a symptom of (heredo) degeneration. Hence we shall be touching on problems, set in the literature, associated with the pathogenesis of disc lesions. Our enquiry, therefore, leads us to an examination of the accepted views on the various possible causes of this degeneration. We shall discuss the various ways in which this morbid change is manifested and at the same time consider what is meant by degeneration of disc tissue where appropriate under the general heading:

Pathogenesis of disc lesions

1. Tumours and inflammation (*e.g.*, sarcoma, tuberculosis and typhoid) may primarily attack the intervertebral disc and even cause it to disappear altogether. But we can find nothing in the literature to show that primary disc degeneration, as understood in this context, may legitimately be considered as neoplasm or the result of inflammation. No pathological changes pointing that way were found in disc tissue removed at autopsy or at operation. We searched diligently for an inflammatory cause of disc degeneration as a systemic disease. No micro-organisms were found It is, however, known that infectious processes (meningitis, epiduritis or post-operative complications of that nature) are capable of affecting the disc secondarily (Krayenbuhl, Lenshoek, Lewin, Prick and Biemond, Schmorl).

2. Degeneration of the disc through strain. Every author, without exception, recognises strain (trauma, strenuous labour) as a possible precipitant of a disc lesion. Some attach only incidental significance to trauma (Biemond), whereas others think that even an anomaly of movement may be responsible for the degeneration of a disc (Junghanns), or the assumption of a forced posture (Thiebaut), or even the assumption by man of the erect posture (Armstrong). Zukschwerdt provides us with a striking example illustrating the effect of heavy manual labour. Scarcely any of the thousands of prisoners who, owing to the war, were very seriously undernourished, either complained or presented the symptoms of disc lesions Once these persons had been liberated, however, and were put to hard manual labour, many of them complained of low back pain and sciatica. This was also the case in those P.O.W. camps in which forced labour was added to undernourishment. Zukschwerdt goes on to say that Lindemann and Kuhlendahl only allow true significance to a trauma if it

affects an already degenerated intervertebral disc.

Pregnancy can be regarded as imposing a physiological strain.

We recognise the following possibilities:

(a) Back complaints during pregnancy are generally to be regarded as physiological disabilities attendant upon pregnancy, temporarily exacerbated either on organic or on psychogenic grounds.

(b) A pre-existent disc lesion worsens: the symptoms rest on an organic basis; the patient retains a "weak back" after delivery; the disc lesion may develop into a manifest hernia nuclei pulposi either before or after confinement.

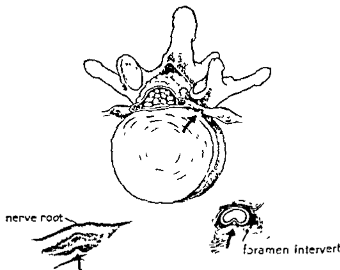
(c) The force of what is no more than physiological pressure upon the vertebral column through pregnancy, combined with that of incidental pressure, will but very rarely be the mechanical cause of the disintegration of a previously healthy disc.

Sicard and Sureau (Spiegel 1956, p. 192) studied the possible relationship between *pregnancy* and a disc lesion. The authors conclude that "the number of pregnancies in each individual patient did not show any definite relationship to the instances of disc hernia" and ". . . . that it appears that pregnancy did not cause any permanent damage to the disc".

It is our personal impression that this statement is too generalised to hold, an opinion which is shared by several authors (Armstrong, p. 147). We know women whose back trouble began for the first time during pregnancy (or delivery) and, after delivery, either ceased or persisted. In several cases a herniated disc had later to be removed surgically. There were other patients, whose pre-existing, indeterminate back pain grew worse. Increased weight, changes in posture (displacement of the centre of gravity, hyperlordosis), hampered movements (less adequate protective mechanisms) will be the mechanical cause of extra pressure upon the vertebral column during pregnancy. We do not know whether hyperaemia and biochemically induced greater extensibility of the tissues have anything to do with it. What we do contend, however, is that a healthy disc will not become diseased on account of these conditions, though a pre-existing disc lesion may worsen and be manifested as the result of this extra pressure.

It will always be difficult to prove that an injury in a given case was the only cause of a lesion and the complaints which seem to result from it. The possible exacerbation of an already existing condition should constantly be borne in mind. The observed fact that a hernia is large or small (Bradford, Briggs), perhaps no bigger than a pin's head (Hanraets), will not help us. Possibly a lateral prolapse is rather more suggestive of a chronic pathogenesis, whereas a disc protrusion in the middel suggests a sudden aetiological precipitant,

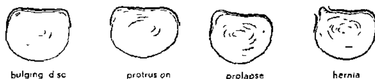
because it might be inferred, both from the vascular distribution in the disc in the earliest years of life and from the scanty expansion of the posterior longitudinal ligament at the postero-lateral side of the intervertebral disc, that this part is more likely to suffer the effects of degeneration (Thurel and



A pin's head "bud" hernia under the nerve root

Zukschwerdt). Soft, maybe necrotic, places in the material of the disc, on the other hand, are strongly suggestive of degeneration, especially if they occur in a collapsed disc. By contrast, the fresh, traumatic hernia will protrude farther, will be engorged and attached to adjacent structures by fibrous tissue. This is an apt moment to recall that Girard questions whether the bulging part which we call a hernia or prolapse derives from the nucleus pulposus at all; he says it is nothing but "une lésion dégénérative du tissu cartilagineux intervertébral".

We describe bulging disc tissue covered by a continuous ligament as a



"*prolapse*", whereas, if this tissue protrudes through a rent in the ligaments, we call it a "*hernia*". In the Dutch literature, a more than physiological bulge of the disc can be equated with a small prolapse. It has been suggested in the German literature that a small bulge of the disc should be called a protrusion

(Junghanns), while they mean the same thing by prolapse and hernia as we do by hernia.

3. Decay of tissue has also been ascribed to: *diminished nourishment of the intervertebral disc owing to changes in the blood supply*. All the authors writing on this matter are agreed that the disc is well supplied with blood during the embryonic stage, but that the blood vessels are obliterated at maturity. (Armstrong, citing Keyes, Bohmig, Chormley, Schmorl, Smith, Übermuth, Watson-Jones). The exact age at which this happens is not known, but it is probably gradual. The disc tissue is nourished through nutritive channels, without an endothelial lining, embedded in the tissue of the intervertebral disc; through these channels lymph is carried from the marrow of the vertebral body *via* the cartilage end-plates to the interior of the disc. According to Armstrong, these channels are also obliterated in the second to third decade, with consequent degeneration of the disc tissue. Harris and MacNab infer from Bohmig's investigations that the disc is arterialised up to the eighth year. Through obliteration of the vessels, apertures are then formed in the tissue, which are later recognised as weak spots and may be the cause of ruptures. (It is for this reason that the disc does not rupture in early life.) Small haemorrhages then occur where there are new ingrowths of blood vessels and where connective tissue is becoming fibrotic. They found these apertures in the tissue in all old discs, including those of persons who had never presented symptoms. These changes – which are also described fully by Schmorl – were found in individuals above the age of 20 and were considered to be quite normal between the ages of 30 and 60. It was this fact which led them to say that they considered a disc to be degenerated if at the age of 30 it looked like one belonging to a person of 60. Their investigation covers 123 routine resections of the vertebral column of patients who had never suffered from back trouble. Following upon this work, they drew up the following hypothesis, which finds support in Junghanns' view, expressed by the term "Bewegungssegment" (segmental unit of movement). They suggested that the vertebral column at the level involved reacts as a whole to this fibrosis – degeneration – of a disc. In their opinion, the damage done to one part of a vertebral segment also produces changes in other structures of the same vertebra. Hence, when a disc collapses due to degenerating, the following changes take place in adjacent structures: fracture or subluxation of the joint, abrasion of the joint facets, fibrosis of the cartilage, lipping as a precursor to osteophytic growth and chondrification or ossification around the joint. Finally, fragments of cartilage may occur as rudimentary joints, while tissue may grow from beneath the attenuated cartilage of the joint surface

right into the bone tissue. The thus collapsing disc is painful; pain is also produced in the muscles, the ligaments and the joints, radicular pain due to irritation of the root, and weakening, necrosis or tearing of the ligaments occurs, through which herniation of the nucleus pulposus may also be precipitated.

To our mind, the foregoing works out as a process, within the context of which the pathogenesis of a variety of pathological conditions in the vertebral column can be clarified.

Seen in this light, it becomes easier to understand why a hernia is so often



found in a postero-lateral localisation. For, the vessels which originally supplied the young disc tissue penetrated dorso-laterally into the disc (Zukschwerdt). After obliteration, it was precisely there that they produced weak spots in the annulus and ligaments. An emerging mass will find it easier to work its way out through this area of least resistance and along the vestiges of the obliterated vessels where they passed the ligaments. Maybe Thurel also had this in mind when he referred to the weaker constitution of dorsal lamellae of the annulus fibrosus. However, we must not lose sight of the fact that probably the dorso-lateral hernia is detected far more commonly because it is more likely at this site to produce symptoms. It is there that the roots run and that the vertebral canal is narrowest; consequently, it is there that a hernia is most likely to cause root compression. Lastly, as has been said, laterally the posterior longitudinal ligament is thinnest, or may be absent altogether, which is tantamount to an open invitation to the nucleus to "pass this way, please"

4. What is known about *changes in the chemical constitution of the disc tissue?* We shall disregard rare biochemical changes producing a specific clinical picture such as ochronosis, which is associated with alkaptonuria (see Epstein; p. 184).

Tissue degeneration of the disc is a phenomenon which, although occurring at all ages, is unmistakably most frequent in the middle-aged and elderly (Armstrong, Krayenbuhl, Schmorl, Zukschwerdt). It is therefore pertinent to enquire whether biochemical changes take place in the disc with advancing age.

On the whole, bradytrophic tissues (whose own metabolism is very low), of which the disc cartilage consists, tend to dehydrate as the individual ages. With advancing age, these tissues are seen to demineralise progressively. According to Burger, ageing is characterised by a slowing-down in biorhesis, which, in other words, is retarded transition of the metabolic processes from a higher to a lower level. Tunbridge says it is typical of the ageing organism that the elastic fibres gradually disappear and the collagen fibres increase by degrees. In contrast to this, Banga and Verzár state that, although elasticity certainly is a function depending on the age of the individual, it is to be understood that the young fibre, not being extensible, soon breaks under slight tension, whereas the older fibre becomes elastic. We have not encountered this standpoint anywhere else in the literature.

We must furthermore point out that, according to Bull, these biochemical senescent changes have nothing to do with any change in nourishment, as has been proved by experiments on animals. Bull noted that, even under conditions of extreme starvation, the body retains its normal chemical composition, broadly speaking. This is compatible with Zukschwerdt's experience, he having, as we may remember, found no increase in discopathy as the result of severe undernourishment.

One of the first references to the water economy of the disc we encountered was in B. Brouwer, who found a lower water content in the degenerated disc. Compère, Keyes, Puschel calculated that the water content of the nucleus pulposus is 88 % at birth ,drops to 80 % at the 18th year and continues steadily to decrease till death to the ultimate figure of 69 %. For the annulus fibrosus they found the corresponding percentages of 78, 70 and 68.

Water exchange in the intervertebral disc is effected by equilibrium between osmotic and hydrostatic pressure; as the result of low osmotic pressure in the disc, water is said to be drawn through the cartilage end-plates from the blood sinuses of the vertebral body into the disc. This diffusion of moisture ceases when the intervertebral body is so saturated with it that the opposing pressure of the surrounding tissues combined is equal to the osmotic pressure (Armstrong). This static pressure is produced by muscular force and ligaments together with the effect of gravity in the erect posture of the body. That is why, during sleep, more moisture is attracted by osmotic force to the intervertebral disc, with the result that 1 or 2 cm (?) is added to the height of the individual on rising, whereas during the day, when the greatest static pressure prevails and moisture is, as it were, squeezed out of the disc, this added height is lost again (De Pukys). The corollary of this phenomenon is that a disc whose spatial boundaries have been damaged by

a lesion will never again be able to keep this game going between osmotic and static pressure. Perhaps this same phenomenon might account for the swelling of a disc to more than its normal thickness as, according to Zukschwerdt, a healthy disc neighbouring a diseased one is sometimes seen to do. In that case the moisture carried from the tissue to the intervertebral body would be available in its entirety to the undamaged disc. It is not considered probable that a disc will rupture on account of this swelling.

Microscopically, too, the *intervertebral disc* changes from birth to senescence. At birth, the nucleus pulposus is mucoid. In the four-years-old child one finds mainly fibrinous tissue and some cartilage cells. At the age of 12 the nucleus consists for the most part of fibrocartilage and a gelatinous matrix and the senile disc is composed of only dense fibrocartilage. In this last period of life, calcification is also found (Armstrong, Krayenbuhl, Lewin, p. 722, von Luschka, Zukschwerdt) Macroscopically, the elderly disc is often fragmented and discoloured to a yellowish brown. Zukschwerdt, however, found this, by no means rarely, in persons of 25, sometimes in several discs at the same time, at others in one only, but invariably more often in the lumbar region than at a higher level. When the elderly *annulus fibrosus* is examined microscopically, the fibres are seen to have swollen and to have reverted towards the hyaline. Macroscopically it is fissured and fibrotic (Krayenbuhl).

The *cartilaginous* end-plates become thinner in later age; they are liable to calcify and even to ossify, the latter applying only to the lateral parts as a rule, since the ventral and dorsal portions are usually not involved.

When changes of this nature are found in the disc of a person aged 25 to 30, it is legitimate, according to Harris and MacNab, to speak of a demonstrable "degeneration" of the intervertebral disc.

The pathological examination of herniated nuclei removed at operation either reveals no abnormalities or else a mucoid and hyaline degeneration of cartilage with pycnosis of the nuclei of the cells.

Hartmann, speaking at the Eighth International Congress on Rheumatism, said he regarded the physiology of tissue as a chemophysical process. If so, the extensibility of tissue could be influenced chemically. He had seen that the effect of hyaluronidase on the intervertebral disc, both *in vivo* and *in vitro*, was to make it less elastic This involves limitation of its function. The splitting of hyaluronic acid, under the influence of hyaluronidase, in the cartilage accounts for the process Water is absorbed but, this notwithstanding, elasticity is first seen to diminish, with the risk of fracturing and tearing, while softening sets in later. The speaker claimed that all this sufficiently explains the occurrence of disc herniation without any specific disease,

because, as the individual ages, changes in pH, heat, the influence of ferments, hormones and so forth provide the conditions necessary for the accretion of the hyaluronidase in an unphysiological degree.

Evers (Third European Congress on Rheumatism, 1955) accounted for the specific effect of sulphur baths for degenerative changes in the spinal column by deposition of sulphur in cartilaginous tissue, which checks the break-down of the hyaluronic acid. De Sèze (speaking at the same Congress), mentioned an experiment in which the hyaluronic acid extracted from intervertebral discs was injected under the periosteum of rabbits, which resulted in osteophytic growth, pathological formation of bone and biochemical changes in the nucleus.

Delaville, Drieux, Lacapère, wondered whether degeneration of the disc might be the result of chemical changes in its tissue in persons predisposed to this at an early age. They had discovered that the effect of hyaluronidase on depolymerisation was far stronger in the discs of very young animals than in very old ones. This would seem to show that biochemical changes in the disc do in fact run parallel with age. Further analysis of the chemical composition of the disc revealed the fact that the total glucosamine content varied from 1.82 % in very young discs to 4 % to 6 % in very old, fresh discs. The glucose content was also found to vary between young and old from 6 % to 9 %.

We may conclude from the foregoing that biochemical changes take place in the disc in step with advancing age. They have also been seen to occur, inexplicably and without symptoms, in youth. The changes observed, however, did not clarify the cause of the process which we call the degeneration of disc tissue. If we accept the term "katabiosis", we could regard these chemical changes as the result of a process of degeneration manifested in the retrogression of the disc tissue. Put differently, although biochemical changes were recorded, these are interpreted rather as a result than a cause of a degenerative process which leads to deterioration of the tissue.

The influence of hormones. Asbol-Hansen (3rd European Congress on Rheumatism, Scheveningen, 1955) reported on his research into the influence of hormones on the metabolism of tissue. (Hydro) cortisone and Acth assist the formation of hyaluronic acid. One object was to ascertain the effect of thyroxine, thyreotropic hormones and the growth hormone on the chemical constitution of tissue, including the moisture content, when it was found that the sex hormones favoured the retention of moisture in particular. The speaker had no hesitation in asserting that the endocrine glands are capable of influencing the chemiphysical balance of the mesenchyme – and thereby its normal functioning – very sensibly within a few hours.

5. *Degeneration of the disc tissue as of stigma degenerationis.* In the following paragraphs we shall mention those authors who, though possibly also recognising other causes of disc lesions, express the conviction that primary degeneration of the disc is a sign of general degeneration.

Magnuson speaks of "degenerated discs" when the entire intervertebral space collapses and the foramen becomes narrower. Larmon, at autopsy, found in ten unselected spinal columns four cases of disc degeneration, one of which was total collapse of the disc without its prolapse.

Girard considers a herniated disc to be a "lésion dégénerative du tissu cartilagineux intervertébral".

Epstein: "Degenerative diseases of the intervertebral disc have long been known" (p. 367). He subscribes to Schmorl's views, adding: "The borderline between the normal and abnormal (in the process of dehydration) (about the third decade) is incident to the wear and tear of living". In saying this, he maintains his view that the degeneration *sui generis* is associated with degeneration under the influence of *predisposing* factors, like dehydration and stress.

Lewin (p. 732) compares a disc to a motor tyre, saying that the latter may have a congenital defect as the result of an error in its construction. "Second-hand discs – due to *degenerative* changes or to injury – are always a risk". He pursues the comparison by equating strain on the back with abnormal wear and tear caused by excessive speeds in a motor-car. He compares the pricking of the disc in lumbar puncture to the "blow out" of a tyre.

Armstrong (p. 36): "The operative findings suggest a process of *degeneration*; the pathological changes are progressive and are degenerative in nature. It is not possible to determine the cause of such degeneration or to say why some discs degenerate and others do not. Wear and tear, trauma, focal sepsis, nutrition and, *perhaps the most important,* the inherent quality of the fibrous and elastic tissue in the individual concerned, may each play its part."

Brocher (p 24) regards degenerative changes as the chief cause of pathological conditions in the spinal column, including degeneration of the disc (influenced by a constitutional hereditary factor, p 30).

Dandy, quoted by Lewin (p 737): "Concealed discs", in the sense of degenerated collapsed discs, constitute about 25 % of the disc lesions.

Cloward, quoted by Spiegel X (p 346), saw 57 *degenerated discs* in a series of 120 patients operated for nuclear retropulsion. "Eleven per cent more multiple ruptured were observed in patients with degenerative discs than in the overall group which the author believes suggests a *constitutional weakness*".

At the Third European Congress for Rheumatism (Scheveningen, 1955) numbers of speakers referred indiscriminately to morbid changes and de-

generation of the intervertebral disc (Barcelé, Evers, Junghanns, von Pap). Several, however, made a distinction by speaking of "degeneration of the *predisposed* disc" (De Sèze, Forestier, Friberg, Rubens-Duval, Vegas).

Zukschwerdt (p. 55), describing the macroscopic and microscopic manifestations of disc degeneration, continues: "so imponiert das Bild doch als 'Degeneration' ".

According to Exner (p. 99), degeneration of the disc is a sign of general degeneration.

Kuhlendahl is of opinion that a trauma will only precipitate a hernia if "durch Degeneration die Bandscheibe unfallsfähig ist geworden".

Let Schmorl have the general last word; the current views on degeneration of the disc and general degeneration derive from the hypothesis he formed years ago. In his description, which has become a classic, Schmorl refers to "chondrosis intervertebralis".

"Das Zusammenwirken der Gewebsaustrocknung mit den täglichen Beanspruchungen (Belastung sowohl wie Beweglichkeit) ist die Ursache für die *Gewebsdegeneration*". He describes the progressive inelasticity and "die braune Degeneration" of the nucleus, the tearing and secondary vascularisation of the annulus and the "Degenerationen in dem Knorpelplatten". He differentiates between *primary chondrosis* (through change and desiccation) and secondary chondrosis (as the result of strain). Either may produce osteochondrosis (if the vertebral body is also involved), "Schmorlsche Knotchen" and hernia nuclei pulposi.

We might say that primary chondrosis is synonymous with degeneration of the disc, the more so because, according to Schmorl, although heavy manual labour plays some part, the *constitutional predisposition is the dominant factor*. Elsewhere (p. 159) Schmorl goes out of his way to differentiate between symptoms of old age, wear and tear and *degeneration*. He says that herniation seldom results from a trauma, but far rather from the accumulated effect of daily strain upon an *already degenerated* disc.

Although detoriotation of the disc tissue may involve a *narrowing of the intervertebral space*, it would be wrong to conclude that every radiologically demonstrable diminution of the disc is the result of its degeneration. For instance, the disc may have been laid down rudimentarily, as it often is under a transitional vertebra. A disc may be lacking altogether, as in the case of a fused vertebra, while there are also intermediate stages, such as the hemi-fused vertebra. It is especially important to remember that a narrow L.V – S.I intervertebral disc may be a congenital defect (Bradford and Spurling, Willis). Further, there are a number of infectious diseases (Schmorl, p. 118) which

may either affect the disc directly, or, indirectly, may cause a localised osteomyelitis in the vicinity of the disc through bacterial metastases. In either way the intervertebral disc may disappear or become thinner, and this may lead to the discovery of an attenuated disc, when the undetected focus of infection has healed.

During routine examination Krayenbuhl found attenuation of the discs in 30 % of the radiographs, but, as against this, in 68 % of a group of patients with back complaints. Learning that Friberg and Hirsch (quoted by Schmorl) found indications of disc degeneration in 40 % to 50 % of radiographs, it is incumbent upon us to allow for these possibilities in our interpretation of these figures, at any rate as far as narrowing is concerned.

In conclusion, let us recall the manifold guises in which degeneration of the disc, taking the form of hernia nuclei pulposi, may reveal itself. Assuming that the extreme forms are known ,we would mention again the small hernia (Bradford and Spurling, Briggs and others) and the minute, bud-shaped hernia (Hanraets) which, if only because of their pointed shape and their wicked localisation – exactly under the root or in the intervertebral foramen –, are liable to produce the most acute radicular pain. As against these, there were the small herniations found in 15 % of routine sections (Andrae, quoted by Bradford, p. 111) and by chance (Alexander) which, owing to their fortunate position in relation to the roots, had not produced pain. Bradford and Spurling surmise that many vague, subclinical complaints must be due to these "subsurgical herniations of the nucleus pulposus". In this they stand by no means alone, as this surmise is shared by several other authors. Seen in this light, and contrary to our comment on the radiological aspects of degeneration of the intervertebral disc, this phenomenon should be credited with even greater pathological significance.

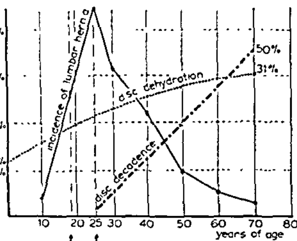
The following points, which we submit as marginal notes, are rather striking:

1 Degeneration of the disc is seen microscopically, macroscopically biochemically and radiologically with progressive frequency in step with increasing age (from roughly 0 % at 20 years to approximately 50 % at 70).

2. The water content of the disc decreases in inverse ratio to advancing age (88 % – 69 %), i e , dehydration increases (12 % to 31 %). If this is plotted in a graph, the frequency of disc degeneration and the degree of desiccation of the disc are found to increase side by side with increasing age.

3. The incidence of lumbar hernia reaches its peak round about the third decade, the onset of symptoms being between the second and third decade. After this, the curve descends steeply (Krayenbuhl, Reischauer, Weber and many others).

From this it follows that herniation is most common in the period of life when the disc *begins* to degenerate and it accounts for the usually negative findings of the pathologist on examining extirpated hernias. Precisely at the age when, by exact methods, the tissue of the disc is found to disintegrate in an increasing degree and, parallel with this, desiccation is at its height, herniation virtually vanishes from the statistics! In other words, dehydration is not the cause of herniation, or dehydration is not a precipitating factor for herniation! On the contrary, it would seem to inhibit herniation. We think, therefore, that the opinion sometimes expressed, to the effect that a reduced moisture content of the disc is responsible for its herniation, is very much open to doubt. A far more plausible interpretation of the curves seems to us to be that the persons in whom the intervertebral disc is degenerating, with the formation of a genuine hernia nuclei pulposi, are *marked off* before attaining middle age; and this looks very like a *predisposition.*



The question now arises as to why degeneration of a disc generally precipitates herniation in early life and does not as a rule in later life. The answer is not to be found in the literature. We might suggest an answer by postulating that when degeneration (determined constitutionally) is already at work in the young disc, it proceeds rapidly under an extreme form, namely nuclear retropulsion; but if the (constitutionally determined) degeneration at a later stage of life is chronic – almost physiological – in character, the disc and adjacent structures have time to adjust themselves and the process therefore follows a milder course. If this is acceptable, it at the same time provides us with a rational relationship between the diminishing water content in later age and the increased frequency of (chronic) degeneration of the disc. For, the decreasing water content of the disc is a physiological process which also takes place in the healthy, ageing disc! We, indeed, incline to the belief that the diminution in water content is not the *cause* of chronic degeneration of the disc in the elderly, but that it very probably is responsible for the *chronic course* of this process. Our reason for this belief is that, while it

is becoming desiccated, the tissue acquires a fibrous, tough structure. It is easy to see that, reasoning in extreme terms, even a calcified fibrous nucleus is less likely to be dislocated than a gelatinous one.

Arguing along these lines, we could say that degeneration of the young disc and of the old is a single process which follows different courses at different ages. Then, apart from the unmistakable effects of trauma and strain, the inference would seem to be that the constitution of the individual determines the age at which and the degree in which the process takes place.

Summing up the views expressed in the literature, it appears to us that "degeneration" of the intervertebral disc running parallel with the age of the individual is generally accepted.

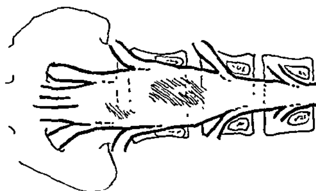
Not a single author, in fact, ignores the possibility, at the least, that premature degeneration of the disc in the early years of life (from no known cause) may be a sign of general degeneration; a constitution predisposing the individual to this process is mentioned several times. Except for the mention of some cases of hernia nuclei pulposi in one family (or even in identical twins – Prick and Hoeberrechts), no explicit opinion is expressed on the possibility of heredity in this matter. In the literature we did find indications, but no proved evidence, of a degenerative constitution predisposing the individual to degeneration of the disc; convincing arguments in favour of heredo-degeneration are not advenced in such cases. This nothwithstanding, the frequent allusions by many authors to the possibility of a constitutional predisposition to degeneration of the intervertebral disc embolden us to believe that this idea has taken hold in the relevant literature.

Contents of the Vertebral Canal
Anomalies of the Dural Sac and Cauda Equina

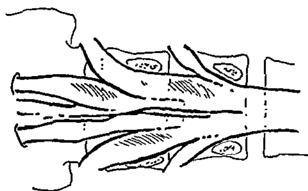
It can be said in a general way that these and other anomalies have often been found in association with developmental disturbances elsewhere. A developmental disturbance, alone, is a rare occurrence, but it should always be remembered that, without complete dissection, it is impossible to be sure that there are no others.

In the literature, low back pain and radicular pain is very often associated with some developmental disorganisation; as against this, however, anomalies have been found in persons who had never presented those symptoms. Hence a causal relationship may possibly exist, but it is not obligatory.

Wide dural sac



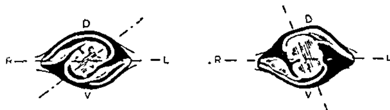
Membrana reuniens persistens. Nerve root anomalies



a. Anomalies of the spinal cord

Actually, these do not belong to the region of the low lumbar spinal column, as the cord does not usually extend beyond the level of L.I – L.II. We shall here disregard these anomalies, as there are meningomyelocele, diastematomyelia, diplomyelia and atresia of the cord with the absolute stenosis as defined by Sarpijener. These, as also the micro-heredo-degenerative signs in clinical cases according to Fuchs and Friedreich, are of interest to us only in so far as they give rise to abnormal mechanisms of movement and to postural anomalies. Within this context they will be discussed in a subsequent chapter.

Two cases are reported of rotation of the spinal cord. The description of these anomalies illustrates how the ganglia may come to be situated ectopically as the result of a developmental disturbance. This was clearly visible in the two cases referred to and its detection provides those who maintain that the ganglia can be drawn out of the foramina by traction (*e.g.* during the ascensus medullae) with strong evidence. Manly (quoted by Epstein, p. 118), reports a



case in which the cord was rotated by about 60 degrees and he noticed that the intradural roots on the left ran over the spinal cord dorsally towards the right-hand intervertebral foramina. The roots on the right, however, crossed the cord ventrally towards the left intervertebral foramina. The ganglia lay in the pathway of these roots against the thoracic cord, so ½ to 1 cm within the spinal canal. Morley describes more than 90° rotation of the spinal cord. In his case, too, the taut roots crossed the cord dorsally and ventrally, with the ganglia in the middle, free inside the canal. The left-hand roots had their origin on the left-hand side of the more than 90° rotated cord and, correspondingly, the right-hand roots on the right-hand side. The latter description seems to us far more plausible than Manly's. His reading of the facts cannot be confirmed without a cross-section of the spinal cord. But it is quite evident from both reports that the ganglia were drawn out of their foramina by traction.

b Anomalies of the dural sac



Many variations in shape and expansion of the dural sac have been observed, some being so pronounced that they could legitimately be called anomalies.

bony rims of the vertebral body together with the physiological bulge of the disc break up the contrast surface and thus simulate these recesses.

In these cases we found a very wide lumbar sac at operation, filling the strikingly wide spinal canal up to the very foramina. The dural sac may be transparent, so that one can see the pulsating intradural roots moving up and down; in these cases the dura was as thin as paper, particularly dorsally. The distension of the lumbar sac reminded us of an inflated toy balloon with a thin place showing. It was stretched dorsalwards and merged laterally, approximately where the roots enter, into dura tissue of normal, almost non-transparent consistency. Consequently, little room was left for the roots in their course; they were pressed against the side wall of the spinal canal, ran alongside the dural sac and disappeared into the foramen at a sharper than normal angle. Through this the bony upper margin of the foramen was able to exert pressure upon the roots. They were therefore compressed in the most lateral part of the spinal canal; at the site of the vertebral foramen this is formed by the sulcus lateralis and, at the site of the intervertebral foramen, by the approach to the foramen (recessus lateralis). We later realised that an oblate wide spinal canal goes with a wide dural sac of this kind and that typically the sulcus and recessus lateralis are particularly flat and narrow; also that, with a wide dural sac, the roots are compressed in this narrowing. We further realised that this predisposed to root irritation; should the available space be further limited by the disc or as the result of arthrosis or spondylosis, the patient would become subject to radicular pain.



A wide lumbar sac does not exclude the simultaneous occurrence of a hernia nuclei pulposi, which has likewise been noted.

It became our considered opinion that the wide dural sac may predispose the subject to symptoms and, maybe, itself produce symptoms through the mechanism described. We were not able to establish a well-defined clinical picture with pathognomonic symptoms. The patients presented vaguely a clinical history and radicular syndrome similar to those encountered with hernia nuclei pulposi. Their movements, however, were not similar to those of patients suffering from a disc lesion; notably, the corkscrew phenomenon was not presented if there were not a simultaneous herniation or disc lesion. In two of the ten cases the patients said that they had an undulating sensation at the base of the back when they *made abrupt movements* on their heels. One patient had this sensation upon suddenly rising from a supine position. To our mind, this is due to changes in hydrostatic pressure, as the result of which the lumbar sac suddenly dilates.

In the last cases of this series, and after their publication (in 1953), the wide lumbar sac was recognised on myelographs and in qualifying cases was accepted as an indication for exploration.

The surgical treatment of the abnormality then consisted in trimming the dural sac to reduce it to more or less normal proportions. In some cases it was reduced to half, or even one third, of the size found at exploration. The aim was to reinforce the dural sac posteriorly and at the same time to pull the roots away from the lateral boundary of the spinal canal. It will be shown in the Chapter on results of surgical treatment, that not only did the patients come through satisfactorily, but their pre-operative symptoms cleared up.



We became convinced that an abnormally wide dural sac *must* be trimmed, not only to clear up the pre-operative symptoms, but also because a wide dural sac with a thin, weak wall is liable to protrude dorsally after exploration (for which the posterior wall of the spinal canal is removed). It is immaterial, we think, whether an arch is left or the arch is removed in its entirety at this explorative operation, because it is even worse if a wide, thin dural sac of this kind begins to protrude dorsally through small apertures between the arches.





A deep dural sac, reaching down to the sacrum is considered to be a variation. We found nothing in the literature to indicate that symptoms are associated with it. Nor is it stated whether a patient who had undergone myelography suffered more from subsequent temporary pain in the coccyx when a lumbar sac reaching very far down had been found.







The shallow dural sac was first mentioned, though not called by that name, by Glorieux in 1937 (p. 57). During a myelographic examination he noticed an anomaly of the lumbar sac: it ended high behind the vertebral body of L.V and divided up, from there, into bundles. The examination was made because of the patient's complaints of low back and radicular pain.

Hanraets (1953) noticed, both myelographically and, later, at the operation, that in two cases the wide lumbar sac ended between L.V and S.I. The sacral

roots were distributed over the extremity of the sac in a way which could better be described as a mop of hair than as a horse's tail. These patients likewise complained of low back and radicular pain. Several identical cases were observed after the publication of the above paper.

Cystic abnormalities. The disorders involving accumulation of fluid in the epidural space are covered by the term *extradural cysts.* They are described as being acquired, but also sometimes as congenital. Differential diagnosis should include colliquative haematoma, the residual condition of an epidural abscess, the epidermoid cyst and the parasitic cyst.

The *post-operative* fluid cyst between the muscles is considered to be a complication after leaving the dura patent, or as the result of leakage when a suture in the dura has not quite closed. But there also appears to be no doubt but that epidural accumulation of moisture may derive from leakage caused by a lumbar puncture.

It seems highly probable that a cystic epidural accumulation of moisture may be one of the after-effects of an *injury.* Twice we found the dura squeezed in a fissure in the L.V arch, one in a person whose clinical history showed a trauma, the other in someone who had not had an injury. A fractured arch may open the dura, when cerebrospinal fluid will enter the epidural space and this fluid would then be secondarily surrounded by a cystic wall.

In 1953 Verbiest described the discovery at re-operation of an ossified cystic wall between L.IV and S.II. During the preceding operation, when nothing unusual had been seen in the epidural space, the dural sac had been left patent, notwithstanding which, no communication was found at this second operation between the dural sac and the cyst which had been formed there secondarily.

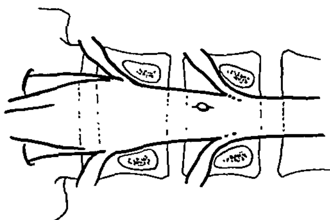


We have seen small apertures in the dura after a trauma, through which several millimetres of the arachnoid protruded. Nosik (1955, p. 2820) demonstrated a similar post-traumatic cleft in the dura myelographically

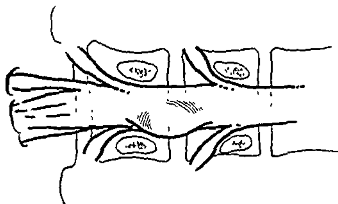
We also once saw a diverticulum, the size of a pea, of the intact dura of L.V – S.I, surrounded by fat tissue and connective tissue adhesions. At the time, we called this an "*abortive" meningocele* Verbiest (1950, p 2429) encountered a similar anomaly together with stenosis of the vertebral canal. Perhaps we were not far from the mark in calling a diverticulum of this kind (abortive) meningocele Under the generic name of "*meningocele occulta*", Epstein describes several guises under which meningocele can occur, *viz.*, meningocele sacralis anterior and meningocele intrasacralis Ingraham speaks of meningocele intraspinalis. The accepted criterion is

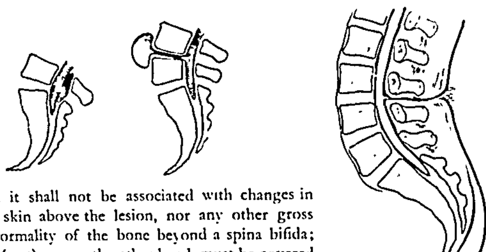


Wide dural sac with arachnoidal diverticulum



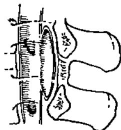
Diverticulum durae (lumbosacralis). (Abortive meningocele)





that it shall not be associated with changes in the skin above the lesion, nor any other gross abnormality of the bone beyond a spina bifida; the (cyst) sac, on the other hand, must be covered with arachnoid and there must be communication between cyst and dural sac. This lesion may be accompanied by anomalies of the spinal cord, sagging of the roots in the meningocele sac, lipoma and sinus pilonidalis. The strain thereby placed upon the filum terminale is liable to induce symptoms. By whatever mechanism, this meningocele occulta is held to produce low back and radicular pain (Epstein, p. 115). Two cases of post-operative "meningocele occulta" after hemilaminectomies were described by Winkler and Powers.

Judging by the many references to it in the literature, *sinus pilonidalis*, or persisting congenital skin sinus, is apparently by no means rare (Epstein, p. 117, Verbiest, "Kinderziekten II", p. 684). Lichtenstein thought its occurrence was a sign of dysrhaphia, due to defective coalescence of embryonic tissue in the median line. This sometimes leaves an open communication between the dural sac and the skin, the external orifice of which may be minute; it is sometimes coloured vascularly or by pigment and long hairs may grow outwards from it. The abnormality certainly does not only occur in the lumbo-sacral region, as several cervico-thoracic localisations have also been described. As this communication remains patent, micro-organisms are able to penetrate into the dural sac, thus entailing the risk of constantly recurring meningitis. Lipomas and epidermoids are found at the intradural end of the communicating passage. Tension in the filum terminale is liable to cause pain and incontinence (Garceau).



It is necessary to distinguish between these, and probably many other, abnormalities of the dural sac and the *congenital extradural sive epidural cyst*. By this is meant a cavity filled with fluid, usually dorsal to the dural sac. In only about one-third of the cases can communication with the intradural fluid space

be shown; according to Epstein, the wall consists of avascular, fibrous tissue lined with epithelial cells. Luyendijk, however, found endothelial cells microscopically, which shows that in his case it was an arachnoidal cyst; the impression one receives from the review of the literature he gives is that the same microscopic evidence was obtained elsewhere as well.

No endothelial cells were found microscopically in the case published by Verbiest (1953). This is interesting in itself, as it had been seen myelographically that contrast medium had penetrated from the dural sac into the cyst. A bulge of the arachnoid is to be inferred from an open communication between the dural sac and a cyst, but the absence of endothelial cells argues against it. The contents usually consist of clear fluid similar in composition to that of the cerebrospinal fluid; rarely is the fluid yellow and gelatinous. Upon myelographic examination, the contrast medium was usually held up; in one third of the cases contrast medium was seen to have penetrated into the cystic sac through a perforation or hole. It is therefore astonishing to learn that on some occasions, when the presence of contrast medium in the cyst had been demonstrated radiographically, no open communication between the dural sac and the cyst was seen at operation. Even on dissecting out in those cases, fluid did not issue from the stump at the site of incision. A valve action is assumed, in an attempt to account for this contradictory evidence, to allow fluid to pass from the dural sac to the cyst, but not *vice versa*. Even so, it is difficult to see why fluid no longer issues from the dural sac once the cyst has been amputated. Valve action comes into operation when, through the abnormality itself, the path of access is opened or closed. Conceivably, the opening of a cyst under great tension is widely patent and closes when the tension is relaxed, *e.g.*, after the cyst has been lanced or removed.

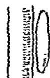
The extradural cyst occurs predominantly in the thoracic region and only very rarely in the cervical and lumbar; Verbiest described the first sacral localisation. Elsberg and co-workers described the radiographical changes, which consist of clearly visible increases in the interpedicular distances in several segments, with erosion of the arches and of the pedicles. The result is a wide, as it were inflated, spinal canal, the dimensions of which have increased, not only in a transverse direction, but chiefly in the antero-posterior direction. These abnormalities are found mainly in young people, especially boys who are X-rayed to find the cause of adolescent kyphosis. The clinical picture is that of the results of medullar compression. The patients complain of numbness in the legs, paraesthesia and disturbed sensation, seldom of incontinence, back pain or radicular pain, but if they do, the radicular pain radiates into the thoracic region.

Spontaneous recovery and remission of symptoms are reported and are attributed to spontaneous rupture of the cyst and to reabsorption of the contents. To this might possibly be added: the coming into operation of the supposed valve mechanism.

Opinions differ as to the origin of these cysts. The general assumption is that the arachnoid protrudes through defects in the dura. If that is so, then the diverticulum and intrasacral meningocele should be classified with these congenital extradural cysts. This is Epstein's opinion and he elaborates it by stating that arachnoidal cyst formation can also be partly intradural. He reports cases of arachnoidal intradural cysts, and extradural cysts, in which the arachnoid caused the formation of a cyst, the site of which was partly intradural and partly extradural (Epstein, p. 447). If this is so, it is difficult to explain why, in two thirds of the established extradural cases, no communication could be found between the dural sac and the cyst, either myelographically or *upon carefully dissecting out at operation*. In those cases it was assumed that the open stump present congenitally was obliterated later on, an assumption based primarily on the fact that an open communication did exist in one third of the extradural cysts.

Hyndman, on the other hand, thought that extradural cysts derive from ectopic cell remains which subsequently swell up and thus produce cysts.

We might therefore speak of *true* congenital cysts of Hyndman and those congenital extradural cysts derived from congenital diverticula of arachnoid and dura.



Several authors have reported on extradural cysts. Schmorl (p. 41) describes the discovery of 48 cystic dilatations in the sacrum at autopsy. He calls these "spina bifida incompleta". These cystic bulges communicated not infrequently with the dural sac, but in the main they were insulated, covered with dura and filled with cerebrospinal fluid.



Glorieux (1937, p. 47) and B. Brouwer (1940, p. 442) assume that symptoms of low back and radicular pain associated with spina bifida are produced by the swelling of any cysts that may be present.

Glorieux describes the myelographic discovery of large cystic abnormalities in the sacrum associated with scoliotic deformities and other congenital defects. Detailed descriptions of extradural cysts are given by Lewin and Epstein.

Orley (p. 338) discusses epidural cysts within the context of the width of the spinal canal. The latest views are set forth by Cuneo (1955) and Burton

be shown; according to Epstein, the wall consists of avascular, fibrous tissue lined with epithelial cells. Luyendijk, however, found endothelial cells microscopically, which shows that in his case it was an arachnoidal cyst; the impression one receives from the review of the literature he gives is that the same microscopic evidence was obtained elsewhere as well.

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had slowly dripped downwards. This experience gives some colour to Tarlov's suggestion that intradural blood slowly seeps along the nerve roots towards the deeper part of the intradural space.

To complete the picture we will add that, during exploration for a sacral cyst of the left S.I root, we removed a "cyst" about the size of a pigeon's egg which later, upon microscopic examination, proved to be a *cystic melanoma.* Fresh blood came from the punctured cyst. When cut through, and after fixation, the tissue was blue-black. In two other cases we found neurosarcomatosis of intradural roots as they entered the root sheath.



Melanoma of S 1 root
(pseudo-sacral cyst)

We do not consider tumours to be one of the possible causes of a nerve-root cyst, but we do wish to point out that, in our own experience, neurosarcoma in the *root sheath* simulates a nerve-root cyst. It may be helpful to know this if one comes across a nerve-root cyst and has to decide what is *to be done*

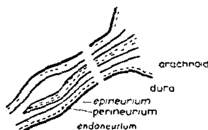
Almost without exception, the *localisation* of the cysts is in the lumbo-sacral region, chiefly in the S.3 root, but they have also been found in the filum terminale.

Tarlov found 5 nerve-root cysts in 30 routine sections and, during a sub-

(1955), the latter reviewing the 34 cases reported in the literature up to that time.

A sharp distinction has to be made between the above extradural cysts and the *nerve-root cyst*, sometimes called the lumbo-sacral perineural cyst. According to Tarlov, who devoted a monograph to the subject, these nerve-root cysts arise between the endoneural plate and the perineural plate of the extradural root sheath or of the capsule of the ganglion. They can be either entirely detached from the ganglion, or united with it as one entity. Cysts may be single or multiple (in about 50% of cases). They may be as small as a grain of barley, shaped like a hazelnut, the size of a pigeon's egg; Mérei found a cyst as big as an apple at the level of S.I.





Tarlov suggests the following causes for the growth of cysts. He is thinking of constitutionally determined cystic decadence of neurogenic elements in the root sheath. The discovery of old blood pigmentation in the walls of many of the cysts led him to believe that the blood derived from a local trauma in the lumbar cavity and, descending therefrom, collected in the perineural space and only secondarily induced the formation of a cyst by colliquation. He found blood, still fresh, in the perineural space of the sacral roots in two patients who had died from a subarachnoidal haemorrhage. Epstein does not agree with Tarlov's idea of cystic degeneration of neurogenic elements, having been unable to confirm this, either microscopically or biochemically, in his material. But Epstein offers no alternative explanation. One does, however, receive the impression that he places nerve-root cysts more or less in the same category as extradural cysts and regards them as arachnoid bulges. Rexed ascribes the origin of cysts to proliferation of arachnoidal tissue in the root sheath.

Our own material supports Tarlov's view. We found old blood in two cysts. During a routine laminectomy for a nuclear retropulsion, by chance necessitating intradural exploration, we saw thin black streaks of blood, several centimetres long, along some intradural roots The blood was so dark that it was thought to come from a haemorrhage several days old. This corresponded to the time when the pre-operative lumbar puncture was made. The blood could not be wiped off the roots and was found still to be under the pellicle surrounding the intradural roots We surmised at the time that the lumbar puncture caused a minor haemorrhage in one or more roots and that this blood

sequent investigation, 8 in 60 routine sections. Schreiber (quoted by Epstein) believes that one case of nerve-root cyst occurs in every hundred operated patients diagnosed as suffering from hernia nuclei pulposi.

Abnormalities are often to be seen in these cases upon careful study of the *plane radiographs*. The anterior sacral foramina are unusually large and the intervertebral canal for the sacral roots may be obviously too wide. Sometimes a posterior concavity is to be seen on the sacrum, where it has made room for the cyst. At operation, very thin bony lamellae are found there.

Nerve-root cysts show up in myelographs. It is said that, in 30% to 40% of the cases, open communication exists between the dural sac and the cyst, so that the contrast medium is able to penetrate into the cavity of the cyst. If the cyst is situated in the median line, it is possible that the lower part of the dural sac is thereby pushed aside, which can be seen myelographically. It has been noticed again and again that, while no indications of a nerve-root cyst appear on myelographs made immediately after the introduction of the contrast medium, it was found to have settled in the cavity of the cyst when check exposures were made some weeks later. Strully does not see in this any reason for accepting the valve mechanism theory. He says that in any event the fluid runs off, under physiological conditions, in the root sheaths via congenitally pre-formed grooves. Thick contrast oil seeps (as does blood of post-traumatic origin) in 16 to 24 days along these grooves into the sacrum; this also happens in the patient confined to bed, because in that position the sacrum is situated lower than the rest of the spinal column. The fact that the accumulated oil (and blood) remains within a cystic invagination of the arachnoid in the root sheath does not in itself imply the existence of a valve action; during the brief period in which the cyst is held at a higher level, the viscous substance is not given a chance to trickle back.

Little of interest has emerged from examination of the *contents* of cysts. The liquid found in them is usually stated to be like the cerebrospinal fluid in composition. The wall of the cyst does not secrete (Strully). It is labour lost to examine this cystic fluid after myelography, because the introduction of contrast medium is bound to have altered its composition.

There is nothing typical about the *clinical picture* of the sacral cyst. The patients suffer from chronic, indeterminate symptoms with remissions. Quite often they complain for years of low back pain or tenderness in the sacrum, with sudden attacks of severe radicular pain which suggest a disc lesion. Then, when a suspect syndrome for hernia nuclei pulposi is not presented at neurological examination (which rarely reveals any neural loss) and when there is no myelographic evidence of herniation, these patients as a rule continue to receive



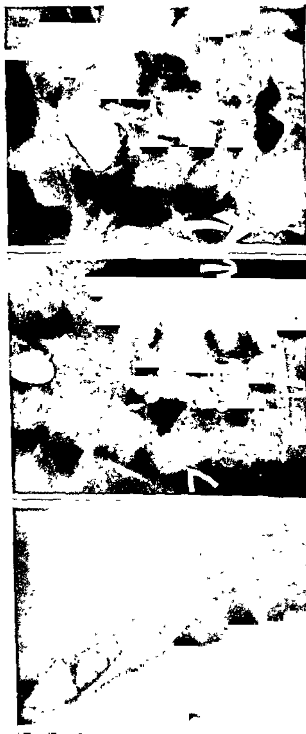


Cyst of the filum terminale, open and trimmed
Solid, thick nerve root S.1
Insipient cystic degeneration of nerve root S.2





Concavities and bony erosion of the sacrum made by perineural cysts



No cranial back-flow of contrast medium in perineural cyst



Myelography of perineural cyst
Visible deformity on plane radiograph
Wide dural sac
Flat oval wide spinal canal



Perineural cyst

conservative treatment, or else an explorative laminectomy (with negative findings) is performed at the level of the L.IV–L.V or L.V–S.I discs. This is not surprising, as the radiating pain produced by cysts of the S.2 and S.3 roots is often vaguely localised by the patient in the whole leg, *e.g.*, the thigh, the latero-posterior part of the calf and the middle underside of the knee. These are patterns of innervation which do not directly suggest those of the deeper sacral roots. If one is not on one's guard, they may easily be mistaken for atypical and inaccurately defined innervation patterns of the L.5 and S.1 roots.

Mention of the nerve-root cyst is occurring increasingly in the literature.

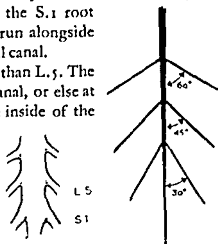
In 1947 Bradford and Spurling (p. 103) expressed doubt as to the existence of the "arachnoidal cyst" of the root. In 1956 Seaman collected sixteen cases from the literature and added five of his own. He gave the names of the authors who had recently written about it. We are inclined to believe that the number he mentions is incorrect, because he gives Tarlov the credit for having been the first to have observed the nerve-root cyst at dissection, whereas we know that Marburg (quoted by Mérei) referred to it as far back as 1902. He says that the nerve-root cyst was first demonstrated myelographically by Schreiber (1951), whereas Glorieux had already reported on several small cysts found myelographically deep in the sacral canal in 1937 (p. 56). Moreover, it is not clear whether Seaman differentiated between the sacral nerve-root cyst and the extradural sacral cyst.

Our own experience extends to a dozen cases verified at operation, including a very exceptional one of at least eight perineural cysts in one patient.



Summing up from Tarlov's monograph it is stated that the perineural cyst is not made up of neoplasm or a dermatoid cyst, nor have signs of inflammation ever been observed in association with it. It should not be confused with an extradural cyst, which is formed as the result of protrusion of the arachnoid through a defect in the dura The cysts arise from the perineurium, predominantly from the S.2 and S.3 roots, sometimes from the filum terminale, and, as a rare exception, have been found at the Th.1 and Th.7 roots. As the cyst is situated dorsal to the root, the sensory fibres are affected sooner than the ventral motor fibres Not only are the cysts multiple in 50% of cases, but they may be locally multi-locular and inter-communicating. The contents consist of clear fluid which is occasionally yellow. The wall of the cyst consists of arachnoidal cells and is laterally bounded by the perineural plate and centrally by more or less necrotic nerve tissue or ganglion cells. The cyst may lie entirely outside the ganglion. No fluid issues, either from the cyst or from the

(extradural) roots. Normally, the L.4 root stands at an angle of about 60° to the dural sac, root L.5 at about 45° and root S.1 at 30°. As a result, the L.4 root crosses the spinal canal obliquely, whereas the S.1 root and the sacral roots situated at a lower level run alongside the dural sac for a longer distance in the spinal canal.



On the whole, the S.1 root is more massive than L.5. The ganglia are situated inside the intervertebral canal, or else at most one third of their girth just comes on the inside of the foramen.



The lowest lumbar roots and S.1 have a certain amount of *mobility*, as has been verified in the cadaver by straight leg raising (Armstrong, p. 31). By comparing this manipulation with the Lasègue manoeuvre, Inman and Saunders, Falconer *et al.* and Charnley saw that the roots had a range of movements from 2–8 mm.

The latter author noted that the root remains stationary while the leg is being raised to 30° to 40° above the horizontal, but from there on it suddenly begins to move until 70° is reached, between which angle and 90° movement diminishes until it ceases altogether.

Sjöquist demonstrated this by showing the film of an operation at a congress held on neurology and neurosurgery in Madrid in 1951.

Woodhall and Hayes have also shown in the cadaver that, when the straight leg is raised, the contralateral roots S.1 and L.5 emerge a little from their foramina into the spinal canal and tend to approach its anterior wall.

Cases are reported in the literature of adhesions, tumours or hernias limiting the mobility of the roots. Under these conditions they may be held so firmly that manipulation, whether digital or by instrument, fails to move them.

Armstrong points out that flexion of the body affects the mobility of the lumbo-sacral roots less than does straight leg raising. We ourselves saw no trace of movement either of the dural sac or of the roots on extreme flexion of the head forwards and backwards during lumbo-sacral laminectomies. A slight displacement of the spinal cord in relation to the dural sac can be seen during cervical laminectomies at a high level. Measuring this photographically, we found that the displacement was not greater than 1 mm.



If, during myelography, a tumour holds up the contrast medium, the level does not change when the patient moves his head in the course of the fluoroscopy. It does, however, if the patient coughs

manifestations of one and the same tendency. Without this postulate, we should flounder in definitions.

We have seen extradural cysts of post-traumatic origin, as a complication after laminectomies and even resulting from lumbar puncture. Apart from these, we know of congenital extradural cysts. Diverticula of the dura and meningocele occulta belong to the same group. What they have in common is a communication, whether demonstrable or not, existing, or having existed, between the dural sac and the cyst. The localisation of the congenital ones is predominantly thoracic and the symptoms, if any, are the result of compression of the spinal cord. During myelography they hold up the contrast medium and the radiograph shows a transverse and dorso-ventral dilatation of the spinal canal with erosion of the arches.

As against these we have the perineural nerve-root cyst, almost entirely confined to the lumbo-sacral region. A genuinely open communication between the lumbar sac and the nerve-root cyst is rare. The swelling shows up on the myelograph if the lumbar sac is low enough to be displaced. In about 20% to 40% the contrast fluid seeps into the cyst. Radiographical manifestations can be observed as swelling of the sacral canal, enlargement of the foramina or excavation of the root canal. The symptoms presented are low back and radicular pain with remissions. Both types of the abnormality may persist for years, producing only minor symptoms, while accidental findings or observations at autopsy prove that they may remain silent. Tarlov and Strully are no more able than Abbot and co-workers to say definitely whether a nerve-root cyst is the cause of complaints and symptoms or not.

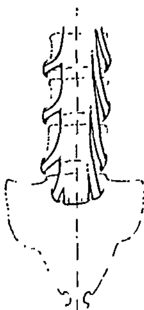
Even in our modest little series of six cases (since increased to twelve) we have a combination of several possibilities, *viz.*,

(a) Free from pain after extirpation of cyst.
(b) Symptoms unabated after extirpation of cyst.
(c) Recurring complaints after extirpation of cyst.
(d) Pain-free after herniatomy and leaving the cyst *in situ*.
(e) Abatement of complaints after herniatomy and leaving the cyst *in situ*.
(f) Pain-free after herniatomy and extirpation of the cyst.

We presume that a perineural nerve-root cyst will produce complaints and symptoms under provocative conditions, such as when a small cyst grows in size or through compression of adjacent roots (if insufficient room is left for these).

c. Anomalies of the roots

By these we mean considerable variations in shape, size and course of the



also be responsible for the fact that the higher roots run obliquely from the lumbar sac to the foramina and that the lowest lumbar and sacral roots tend rather to lie vertically beside the dural sac. (This has been investigated by Barry and co-workers (p. 292) in the human foetus.) One is therefore apt to think of "traction", *e.g.*, tension in the roots between the foramina and the dural sac. We have to keep these mechanisms in mind to account for symptoms produced by anomalies of the roots.

Anomaly of shape through insertion at a high level. The simplest anomaly here is when the lumbo-sacral roots lie almost vertically alongside the lumbar sac. This is considered to be a variation. Thurel says that in 10% the S.1 roots emerge from the dural sac at a higher level than usual; normally the insertion of S.1 in the dural sac is just above the L.V – S.I disc, whereas with the above variation it is inserted just under the L.IV – L.V disc. Consequently these roots lie beside the dural sac before they disappear into their foramina.





Unsymmetrical insertion of nerve roots

by request, but this involves an entirely different mechanism, as the sudden rise in pressure displaces the spinal cord plus the tumour (and the halted contrast medium) relative to the dural sac, causing pain of varying severity. If it is a case of a tumour attached to the dura (meningeoma), which has made an impression in the spinal cord, it is mechanically impossible for the cord in the dural sac to move up and down as the result of movements of the body. The same applies to severe arachnoiditis with strong local adhesions, as in syringomyelia.

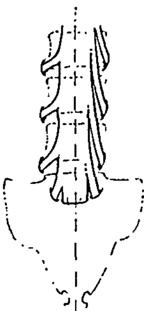
Apart from all this, it may safely be assumed that firm fixation of the spinal cord to the dural sac is ensured by the ligamentum denticulatum.

We are persuaded, by our own observations at operation and by the above reasoning, that the spinal cord scarcely moves at all in the dural sac in response to movements of the body. The same arguments do not hold for the mobility of the dural sac plus contents in relation to the spinal canal. For, its mobility is greater and, thinking in mechanical terms, one might represent the dural sac as being able to move up and down slightly in the spinal canal, checked only by a few fibrous connections and by the roots in their extradural course. When verifying movements at operation, it seemed as though the mobility of the lower part of the dural sac was due to some yielding coming from a lumbo-sacral direction; the impression one receives is that the lumbar sac higher up in the spinal canal is stationary and that all the mobility of the lower part has to be effected by the "giving", shifting or stretching of the lumbo-sacral roots. This implies that limitation of mobility through, say, a tumour, hernia or inflammation localised in the lumbo-sacral region, is more likely to bring on symptoms through movement, because the greatest amplitude of movement is just there. This greater amplitude of the lumbo-sacral range of movement might be regarded as a result of the mechanism described as the ascensus medullae. Tarlov expresses its influence upon the length of the roots in millimetres

C 1	3 mm
C 8	27 mm
Th 12	81 mm
L 4	151 mm
L 5	170 mm
S.1	185 mm
Co 1	266 mm

The cervical ganglia are situated centrally in their intervertebral canal.

Zukschwerdt postulates that the *lumbar ganglia* are drawn somewhat farther into the spinal canal by the ascensus medullae. If that is so, the ascensus may



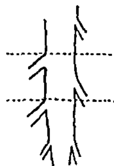
also be responsible for the fact that the higher roots run obliquely from the lumbar sac to the foramina and that the lowest lumbar and sacral roots tend rather to lie vertically beside the dural sac. (This has been investigated by Barry and co-workers (p. 292) in the human foetus.) One is therefore apt to think of "traction", *e.g.*, tension in the roots between the foramina and the dural sac. We have to keep these mechanisms in mind to account for symptoms produced by anomalies of the roots.

Anomaly of shape through insertion at a high level. The simplest anomaly here is when the lumbo-sacral roots lie almost vertically alongside the lumbar sac. This is considered to be a variation. Thurel says that in 10% the S.1 roots emerge from the dural sac at a higher level than usual; normally the insertion of S.1 in the dural sac is just above the L.V – S.I disc, whereas with the above variation it is inserted just under the L.IV – L.V disc. Consequently these roots lie beside the dural sac before they disappear into their foramina.





Unsymmetrical insertion of nerve roots

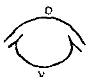


Thurel points out that these possibilities have to be borne in mind, since the L.5 roots are correspondingly inserted a little higher up; therefore, when exploring at the level of L.IV – L.V, the L.5 root might be mistaken for the L.4 root. We have also come across this in even more extreme forms. We shall report some of the cases.

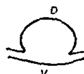
Unsymmetrical insertion of the roots. It has been found (Hanraets, 1953) that in some cases the roots rose alternately from the dural sac. *E.g.*, in some cases the L.5 root was situated at a level between the exits of the contralateral L.5 and S.1 roots.



Mop. In two cases of a very dilated dural sac ending at the level of L.V and S.I, the L.5, S.1, S.2 and S.3, etc. roots were seen (Hanraets, 1953) to be implanted closely packed together in the lower part of the dural sac. The site of insertion was in the dorsal part of the dural sac.



Dorsal insertion (Hanraets, 1953). In a wide, not shallow dural sac the roots have also been seen to rise from its dorsal part. In this case, therefore, the dural sac was distended on the ventral side.



Ventral insertion. In a dilated dural sac we saw a swelling chiefly of the dorsal part, the roots being implanted ventrally, close together.



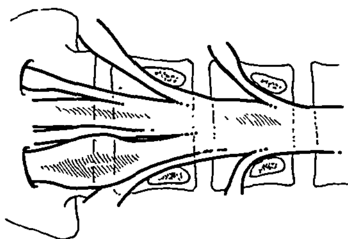
Angulated course of the roots in a dilated dural sac. The usual picture presented by the dilated lumbar sac is one in which the roots are forced by its shape against the lateral boundary of the spinal canal. They then run briefly alongside the dural sac, thereafter passing, at a smaller angle than the normal one, into the foramina (Hanraets, 1953).



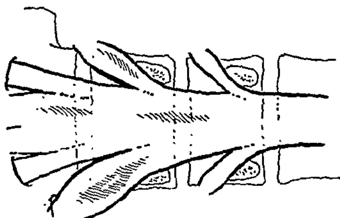


Division of one stem into two roots. Thurel describes the anomaly of two roots, close together, leaving the dural sac. He saw this in the L.5 and S.1 as well as in the L.4 and L.5 roots. He added that the common stem of such an anomaly was compressed by a hernia nuclei pulposi. Hanraets has also described this anomaly in the L.5 and S.1 roots. In the case he was referring to – subsequently followed by others (including this anomaly in the L 4 and L.5 roots), of which photographs were taken – intradural exploration revealed that the intradural roots ran jointly towards

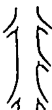
Ectopic ganglion



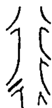
Abnormal girth of nerve root



one exit. There was another instance in which the intradural roots for L.5 and S.1 ran towards two different openings in the dural sac with an intervening distance of only 2 mm. Outwardly, only one thick common root sheath was to be seen.*



Absence of a root. In 1953 Hanraets found that no root emerged from the dural sac at the level where the L.5 root is normally found. In such a case the root situated at one segment lower down was found to be abnormally thick. It was surmised that the nerve fibres which ordinarily pass through the root now wanting had found a path through a root sheath situated at a lower level.



In 1956 Alexander published a case of lumbar myelomeningocele combined with hernia nuclei pulposi and complaints of incontinence. It was discovered at operation that there was no left L.5 root, but the left S.1 root was cleft and, as a whole, was thicker than it normally is. This reminds us that Magnuson (1944) found in his dissecting material a root at the exit of the intervertebral canal which was split in two by a pointed edge of bone. Possibly, therefore, this is a similar phenomenon to that which we saw at foraminotomy, when roots from a common stem divided again at the exit of the canal.





Curled root. Verbiest (1953) reports a case of exploration for the cause of radicular pain when, at myelography, the contrast medium was arrested at the level of L.IV. When no disc lesion was found at operation, he explored intradurally and found an intradural root twisted into a curl like that of a pig's tail. After the adhesions around it had been removed, the radicular pain subsequently ceased. Upon palpation of the dura, the curl could be felt, like an elastic nodule. We came across something similar. It was an intradural root which entered the sheath of the L.5 root in the normal manner and, after turning sharply on emerging, returned, only to disappear at a lower level in the sheath of the S.1 root.



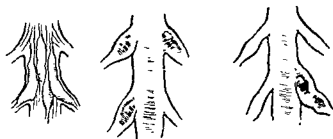
Abnormal girth of the roots. Abnormal girth of the roots was mentioned more than once in the foregoing considerations of anomalies of shape. Thus we have encountered reversals of the normal proportions, in that the L.5 roots were far thicker than the S.1 roots. We postulated that in such cases the fibres appropriate to root sheath S.1 passed through the L.5 root sheath.

* Bolk dissected a metameric division of roots in the shark. Contrary to the usual anatomy, several roots sprang via a common stem from the dural sac (personal communication of one of his pupils).



Small differences were not considered to be an anomaly, but in the above cases L.5 was about twice its normal thickness, whereas S.1 root was reduced to a mere thin strand. The question now at once arises as to whether an abnormally thick root is to be ascribed to an anomaly, since several investigators mention oedema of the roots (Krischek, Voris, Zukschwerdt), especially in the French literature (Cossa, Thurel).

We have performed rhizotomy in the root sheaths of these abnormally distended roots. No signs of inflammation were seen macroscopically in small fragments of root parts removed at rhizotomy. Changes in the girth of the root must be due either to the *growth of a tumour*, accu-

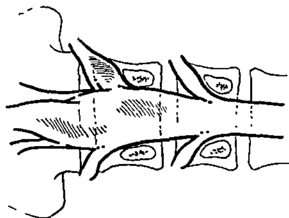


mulations of fluid or anomalies. Twice we found a neurosarcoma of the intradural root, which had extended through the root to the root sheath, which it invaded Thickening of the root due to *accumulation of fluid* is easily recognised by the open communication which exists between the root sheath and the dural sac. The root sheath can be emptied by squeezing and, after an injection – say, with Procaine –, this runs straight into the arachnoidal space and the root does not swell up.

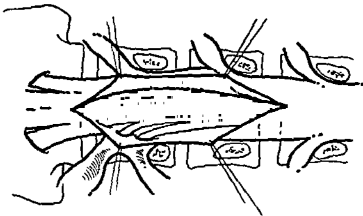
Although we cannot deny the existence of oedema in the roots, we have very rarely come across it at operation or upon microscopy of parts removed during rhizotomy. Distension of the roots as an *anomaly* may occur in one root only, bilaterally or in different roots at several levels. We only once encountered this last. Finally, an abnormally placed ganglion may be responsible for the distension, even though its macroscopical aspect does not suggest that *ganglion tissue is situated in the sheath of the dura*. Very much to our surprise, microscopy of material obtained during rhizotomies performed in the root sheath revealed the presence of ganglion tissue, for nothing in the appearance of the abnormally thick root suggested anything of the kind.

We consider the abnormal route of the intradural root fibres through sheaths alien to them as being the most common cause of an abnormally distended epidural root.

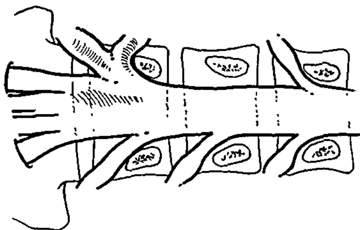
Cystic nerve root (with a wide dural sac)



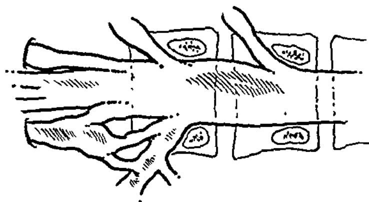
Nerve root anomaly (division of one stem into two nerve roots) Intradural



Nerve root anomaly (division of one stem into two nerve roots). Extradural



Nerve root anomalies and an ectopic ganglion. Trimmed dural sac



d. Anomalies of the ganglia

It has already been stated that, through rotation of the spinal cord, the ganglia may be pulled out of their foramina. Zukschwerdt believes that the ganglia shift, relative to the intervertebral canal, towards the vertebral canal as the result of traction during the ascensus medullae. It has been observed in dissecting material and at operation that the lumbo-sacral roots are mobile, which means that the ganglia attached to them likewise move. We have noticed during operations that the ganglion is not always within the intervertebral canal; we saw it lying close to the dural sac on several occasions, and once it was squeezed into the intervertebral foramen. When the ganglion was closely contiguous to the dural sac, we saw fibrous adhesions. In one case these were so formidable that the S.2 root had fused with the ganglion of the S.1 root, the whole of which was inside the spinal canal (Hanraets, 1953).





In another case we encountered a ganglion of the right S.1 root placed between roots L.5 and S.1; bundles of nerves ran between these two roots. These *ectopic ganglia* were on the whole thicker than usually found in association with such roots. We do not know whether there is any question of oedema in a case of this kind, nor whether such a ganglion contains more than the normal quantity of cells. Ectopic ganglia sometimes coalesce, not only with the dural sac and an adjacent root, but also with all the other adjacent structures and are barely to be moved We have also seen them embedded, as it were, in the bone.



CHAPTER 4

CONSTITUTION IN A GENERAL SENSE

"Constitution" is a rather vaguely defined word and, as generally applied, has various connotations. Before we proceed, therefore, we would do well to explain the sense in which we use it.

To us, then, *constitution* is the genotypical, endogenously determined state of body and mind. In this sense we take it to be the aggregate of genotypical dispositions, hence the complex of the genotypically determined disposition.

In medical parlance, however, the word constitution is also used (to our mind erroneously) in relation to a variety of disorders which, upon closer scrutiny, prove to be wholly or partly acquired, either pre-natally (*e.g.*, congenital syphilis) or post-natally (*e g*, enfeeblement and change due to tuberculosis or a trauma), the cause of which would seem to be entirely exogenous. The endogenous predisposition to the contraction of a particular disease initiated by an exogenous agency is, after all, a recognised fact. The disease (tuberculosis) – the result of an exogenous agency (the tubercle bacillus) – may then be said to be predetermined by a constitutional factor (the susceptibility to tuberculosis) and produces an acquired constitution (the changes resulting from the ravages of the disease).

This is the acquired state of being which we call the *condition*. The condition is the ever-changing total state of being of body and mind, resulting from the constitutional state and from the day-to-day changes incident upon life

In the ensuing pages, therefore, we differentiate, where possible, between:
the constitution = the endogenously determined *state of being* and
the condition = the acquired, variable state of being *modified by day-to-day life*.

Then we have *disposition*, which could be equated with "sensitivity to" or "susceptibility to" The object would be a given injurious agent or influence which, of course, must be present and have time and opportunity to impinge upon the subject, otherwise the subject's predisposition would continue to lie dormant. (A person of delicate build, who becomes over-fatigued by the least exertion, can avoid distress and discomfort by leading a quiet, placid life.)

A *cause* precedes an effect, in accordance with the metaphysical postulate that there can be no effect without a cause. If an effect is observed, there must, *ipso facto*, be a cause, even if this be not demonstrable. Neither a "constitution", itself, nor a "condition", itself, can ever be the cause of anything. It is the interplay between the constitution and the condition that enables a cause to take effect.

There is also *adaptation*, which is a tendency to correct an existing condition to a subsequent condition under the influence of changes in the environment. Hence, in the whole man, it is a vital event which, through adequate psychobiological changes within the organism, subordinates the external situations evoking the adaptation, to the organism, for the benefit of his optimum self-realisation (Prick). If, for example, a human being is exposed in his environment to tubercle bacilli, his system will be compelled to enlist protective mechanisms. However, these recruited processes, which imply man's reaction to the object threatening him, ultimately subdue the invader and the individual's optimum self-realisation is thus salvaged. From this it will be clear that adaptation is always a struggle for life.

Self-realisation is the actualisation, with mobilised intrinsic vital forces, of the endogenously given dispositions in conformity with the immanent law of development (Prick, 1958).

An exogenous factor is one brought about by an extraneous influence. It therefore qualifies a relationship between the medium and the influence. "Exogenous", as applied to human beings, derives its meaning not only from biological, but also, and more especially, from psychological values, both depending supremely on the individual's experience in early childhood.

An endogenous imperative proceeds from the genotypical pattern of the individual. Endogenous and constitutional may be taken as synonymous concepts, provided it be understood that the use of the word "constitutional" implies emphasis on a complex of dispositions, whereas "endogenous" implies the genotypical determination of a disposition. Strictly speaking, therefore, the expression "endogenously constitutional" is not tautological. It is misleading to speak of an "endogenous cause" when the meaning it is intended to convey is "endogenously vulnerable to the effects of a cause".

The diseases referred to in common parlance as endogenous will as a rule be of an endogenous – exogenous character. (Sacralisation, determined endogenously, disposes the individual to suffer from the effects of imposed strain; the degenerative back predisposes to weakened resistance to exogenous factors which produce symptoms.) The vast majority of the so-called exogenous disorders in daily practice are in fact endo-exogenous in nature. (A trauma

which will produce symptoms in someone suffering from spondylolysis need not necessarily do so in another with a normally constructed spinal column.)

A disease or disposition is *congenital* if present at birth.

Hereditary may mean either "brought about by heredity", or 'heritable by descendants". Anything endogenous is, by definition, existent by heredity, but it does not imply that the same characters will be transmitted to the offspring.

Degeneration in its general sense means: going to ruin, disappearance or decline of morphological and functional properties of tissue or organic systems. In the vernacular, "degeneracy" and "decadence" are used indiscriminately.

In this study we use the word "*degeneration*" to denote a (degenerative) process of development in a declining sense, liable, for instance, to lead to retrogression or disintegration of tissue. Hence a *degenerative constitution* is a complex of the genotypically – thus hereditarily – determined disposition as the result of a declining process of development.

We use the term *degenerative back* to denote the manifestations of such a disposition in the various organs of which the human back is composed, disturbed function of what may appear to be normal organs, as well as the pathologically premature onset of otherwise physiological processes of wear and tear.

Constitutional diseases. This is a term which is liable to give rise to misunderstanding. An analysis of these possible misunderstandings reveals a remarkably clear picture of what the genotypical constitution really is. We have found, upon enquiry, that many a doctor regards as constitutional diseases those brought on by – *i.e., as the result of* – the constitution. We, on the contrary, are inclined to believe that these diseases are contracted *in spite of* the constitution. An inviolate constitution that has not yet deteriorated through bad influences into an inferior state of being should hold off illness. If the body proves to be ill, from no demonstrable cause, we have the option (a) to consider whether a cause, though not yet demonstrable, actually exists, or (b) to assume that *the constitution itself is diseased.*

It is necessary to invoke philosophical concepts to bring some clarity into this obscure statement. A "predisposition to something" is fictitious. This concept derives, not from the individual appearing to be predisposed, but from the mind of the observer at the moment when he becomes aware of the result of this supposed predisposition. He names the observed phenomenon and, rightly assuming that it was present *primo originis, calls* that presence at the outset *the predisposition* of the subject Hence the aggregate of the observed

facts *is*, to him, the patient's predisposition. The mode of manifestation in the first instance (whether observable or not) and the phenomena observed in course of time are modulations of one and the same theme.

The genes are credited with the power to modulate. How this power comes into effect, we do not know; nor does it primarily concern us here. It might materialise through electrical discharges, or, according to the latest ideas, through chemical processes. If we accept the latter hypothesis, we must admit that, by producing minute quantities of gene hormones, the genes have the power to form the body (and the psyche). If these gene hormones do this by making use of a relay (generating and utilising glands with internal secretion), then this secretion is the gene hormone, no more no less, seen under a magnifying glass. Disregarding external influences, the same potency *is* present; it can therefore be said that the sum of the potencies thus modulated into materialisation in the further developed individual, undisturbed by external factors, *is* his or her predisposition.

If we see a given likeness between father and son, such as obvious acromegaly, we say that the son was born with a predisposition to the disease. Let us put it simply, thus: a given growth is the result of a growth hormone; the presence of growth hormone in large quantity in the son's blood *is* the predisposition he had inherited and is not the result of that presence.

If the modulation of the potentially present predisposition is disturbed in the developing individual, it is:

(a) either the result of a factor external to the disposition (*e.g.*, X-rays on a fertilised ovum cause deformities or death);

(b) or disorganisation of the modulation itself, which means to say that disruption of the disposition was inherent in the disposition itself.

Put differently, the disposition itself is disrupted, or, in other words, "the constitution is diseased".

In this sense we can accept the expression "constitutional disease".

The manifestations of these apparently causeless diseases represent the disturbed constitution. Such manifestations may, moreover, be revealed in, say, biochemical changes. It will not always be possible to classify these biochemical (etc.) changes into primary properties (= manifestations) of a disturbed constitution and into secondary externalisation which is the logical progression (= result) from the primary manifestations. For example: diabetes – determined constitutionally – may be called a constitutional disease, provided no pathological cause can be adduced. The destruction of Langerhans' islets could be regarded as the primary manifestations of a disrupted constitution; *i.e.*, the diseased constitution itself. This is allowable, provided there be

no convincing evidence suggesting primary disturbances of function in the hypophysis or hypothalamus.

Hypo-insulinism and hyperglycaemia are dubiously primary (native to the constitution). The reduced glycogen content of the liver, thirst and, for example, over-susceptibility to infection are undoubtedly secondary, *i.e.*, effects of the disease (and are appropriate to the condition brought about by the disease).

Constitutional diseases are liable to progress dynamically, with clearly marked changes (leucocytes in the case of leukaemia), but, alternatively, they may be static in character, the data being difficult to establish by current techniques in the laboratory. (*E.g.*, asthenia: fibrogenesis imperfecta).

A "constitutional disease" may affect:

systems	diseases of the spinal cord: syringomyelia?
	diseases of the blood: leukaemia.
organs	pancreas: diabetes,
	hypophysis: acromegaly.
cells	reticulosis
	neoplasms??
tissues	interstitial tissue } asthenia distrophia musc. progress.
	muscle tissue }

Proceeding from this premise, we might come to see the many and various ways in which the degenerative back expresses itself – or combinations of these ways – as just as many manifestations of constitutional diseases. This would mean that spina bifida occulta, the anomaly of the cauda, spondylolisthesis, the – not purely traumatic – disc lesion, and so forth, are manifestations of a diseased constitution. In other words, the degenerative back is not the result of, but represents, *i.e.*, *is*, an expression of the disturbed disposition.

Availing ourselves of the concepts defined in the preceding paragraphs, we shall briefly recapitulate our trend of thought and, where necessary, amplify it.

We have, then, broadly, the constitution, which is endogenous, and the condition, which is endo-exogenous. The realisation of these dispositions is effected, not only by endogenous (hereditary) factors, but partly also by exogenous influences. If the nature of the exogenous factors comports with the endogenous disposition, the realisation of this disposition may be very marked. But if the nature of the exogenous influences conflicts with that of the influences to which the subject is endogenously predisposed, the assertion of hereditary predisposition may be minimal.

It follows from this that endogenous and exogenous factors cannot be dissociated. Exogenous factors may either further or oppose the actuation of

endogenous factors; in like manner, endogenous factors may strengthen or weaken the effect of exogenous influences. The actual total state of an individual at any one moment is the complex of realisations effected as the resultant of the dialectical relations between endogenous and exogenous factors. Now the total state of being is dynamic. We can well imagine that acquired characters, which are integrating elements in a "condition", might be effaced, or disappear, in a later phase of life. Thus one can later lose an actively acquired immunity to some particular agent of disease, that immunity having at a previous time been an integrating factor of an existing condition. Losing this immunity, the individual enters, at that moment and by that fact, into a different condition.

We asked whether a labile constitution or condition can be said to be a cause of diseases and came to the conclusion that it could not. The cause of a disease is an exogenous factor (or complex of exogenous factors) which, by virtue of its intrinsic nature, is capable of inducing a disease. A given factor can only be claimed as the cause of a given disease when satisfactory evidence has been adduced to show that this particular disease never occurs in the absence of this particular factor. The cause of a disease is that which is the *sine qua non* for the occurrence of that disease. All other factors, whether extraneous or present within the organism, which are incapable of inducing the disease by virtue of their intrinsic nature, are merely conditional.

These conditional factors can be subdivided into predisposing and precipitating factors.

The precipitating cause of a disease need not be of like intensity in all concrete cases; this is where the influence of the predisposing and precipitating condititonal factors comes in. If the individual is endogenously predisposed to contract the disease, *i.e.*, if, through his or her hereditary make-up, morphological or functional conditions have been realised which favour or impede the onset of the pathological process, the precipitant cause can be less or more intense, depending on the condition realised. It is evident from the foregoing that neither the (endogenous) constitution, nor the (endo-exogenous) condition can legitimately be stated to be the cause of a disease; they are predisposing pathogenic factors.

Precipitating factors, which should be placed side by side with the predisposing ones, are extraneous influences, *in*capable, of themselves, of producing the disease but which, on impinging upon the organism, bring about certain conditions favourable to the precipitating influences of the actual causes of the disease. Let us clarify this by an example. The true cause (causa efficiens) of gastric ulcers is unknown; but it is an established fact that a well-defined endo-exogenous condition in man bears the signature of a

associated with their anomaly. In our opinion, admissible conclusions cannot be drawn from this large-scale investigation – the only one we know of – into the relationship between symptoms and anomalies.

Van Assen (Sleeswijk, p. 561) ascribes the presentation of symptoms in middle age, produced as the result of deformities present at birth, to deterioration in the functioning of the muscular system, which was adequate in youth.

Others (Brocher, pp. 24 and 26) lay more stress on the resistance governed by the constitution, saying that it takes a severe strain to injure the tissues of a youthful person, whereas no more than a physiological strain may do so in the same individual later in life.

Martius (quoted by Ruding) says that, considering that man slowly acquires the habitual automatic posture, which does not become established until the years of development are past, it is not surprising that the symptoms referred to should not occur until the second decade.

Bistrom found 69 lumbo-sacrally localised anomalies of a varying nature in 151 persons between the ages of 30 and 70, yet none had ever had back trouble, despite hard work.

Holt, Hodges and co-workers, in whose Year Book of Radiology we found a report of Bistrom's investigation, subscribe to the conclusion drawn from it. They point out that well-founded investigations of the above type have been published during the past twenty years, the results of which are a warning to radiologists and orthopaedic surgeons to beware of attaching value to the significance of congenital anomalies.

Pugh (quoted by Brailsford) found deformities in 85% of the backs he examined, inferring from this that the majority of these deformities have no clinical significance.

Willis (quoted by Southworth and co-workers) says that, after all that has gone before, hernia and congenital anomalies (to account for low back pain) "are now in fashion".

Schaap (p. 3356) sees no connection between the structure of the spinal column and the occurrence of low back and radicular pain.

Zukschwerdt (p. 69) cites the following pithy definitions:

Disposition = wechselnde Bereitschaft zur Krankheit.

Konstitution = auf Vererbung und Erleben beruhende Korperverfassung.

The impression one receives is that Zukschwerdt does not countenance the "congenital or constitutional predisposition" to morbid changes, at any rate as far as the pathology of the small joints is concerned.

By far the majority of authors attach *great importance to* the constitution

borne, or refer the incidence of complaints to it, at least partly.

Contrary to Pugh (see above), others advance the frequent occurrence of deformities as an argument in favour of the influence of the constitution.

Gutman (Congress Freudenstadt 1956) stated that in 95% of cases the adult spinal column is not "normal".

Rasch (same Congress) observed "abnormalities" in 60% to 80% of school-children. Not all these abnormalities, deformities or variations call for treatment. They are to be considered in the light of Bohart's quotation (Schmorl, p. 253): "Die gesamte Wirbelsäule ist ebenso individuell wie das Gesicht jedes einzelnen Menschen". Hoelen means the same thing when he refers to the "lumbo-sacral anarchy".

Schmorl entertains the greatest respect for the constitution in general and for that of the spinal column in particular. Being aware of this, we have appealed to him several times in the foregoing. ("Der Konstitution ist die grossere Bedeutung zuzumessen").

Lewin (p. 54) does not expect the influence of the constitution and of anomalies of the lumbo-sacral spinal column to make itself felt until the individual is past 40.

Lewin (p. 255) describes the condition of a person whom he regards as predisposed to present a "hyperacute locked back syndrome" in the following words: "The type of person vulnerable to these attacks is usually the over-worked man between forty-five and fifty-five years of age, who for years has been under mental and physical strain. He has discontinued all athletics and sports, occasionally 'kicks over the traces', goes to a cocktail party and dance. He is usually overweight, with a low basal metabolism. He eats too much purine-forming food and is practically 'gout imminent', he has previously had several attacks of lumbago, subacromial bursitis and painful feet. He is usually a 'heel walker' who has been guilty of dietary and other indiscretions."

Kretschmer's classification of types of constitution is well known.

Sheldon is among the champions of the idea of constitution in the sense of disposition in individual development. He notes strikingly common dysplasia in the development of the muscle, joint and tissue apparatus among several of the types he formulates. Investigators familiar with this subject know that this dysplasia constitutes pre-eminently a disposition towards the development of low back and radicular pain. Sheldon goes so far as to say about one type (p. 190): "Women who are 4.1.5's" (*i.e.*, a certain blend of the endo- ecto- and mesomorphic type) "are inclined to have *weak* backs".

Upon the conclusion of an investigation into constitution by means of Sheldon's anthroposcopic/somatotype test, D. Brouwer (1956) expresses it

as his opinion that it is not yet possible to study the old problem of "body build and psyche" by accurate methods. His enquiry into the relationship between body build and disease did not produce convincing evidence in a positive sense, contrary to the conclusions arrived at by other authors, like Draper, Parnell, Catch and Eertmark (see Brouwer).

Veil and Sturm (p. 429) conclude thus: "Im Zwischenhirn sind unseres Schicksals Sterne". "Die konstitutionell hereditaren Bedingungen fur die Sphare des Zentralnervensystems reichen noch viel weiter".

In his inaugural address, Van den Bergh (p. 28) expresses the opinion that future medical research will be largely devoted to the study of the constitution.

Reischauer (p. 2) found that the 4% of his patients suffering from back ache for the first time between the ages of 10 and 20 were all young girls with a hypoplastic constitution.

Brocher (1957) described in a monograph the predisposing constitution to low back pain and radicular pain. The prognosis for middle age is unfavourable above all when degenerative changes are found in the spinal columns of young people, even if they present no symptoms.

Let it be said by the way that the constitution is believed to be in part responsible for many diseases of a number of organs besides the spinal column, to which we have here been paying special attention. (See: J. Bauer: *The Person Behind the Diseases*, 1956). Here are two very diverse examples: the pathological conditioning to neuro-psychic disturbances (Prick 1957) and the hypersensitivity to metals (lead) as the result of a degenerative constitution (Marlet, p. 866)

Constitution as associated with the erect posture of man

In the opening paragraph of his Preface, Armstrong writes: "When, long ago . . man forsook the trees to walk upright on the ground, he made novel demands on his lumbo-sacral spine It may be that the process of adaptation is still incomplete". On page 34 he says: "It is possible that degeneration of the nuclei of the intervertebral discs belongs to that considerable group of lesions, semi-mechanical in origin, which constitute one of the penalties paid by man for the assumption of the upright position".

As far back as 1925 and 1928 Harrenstein, in the Dutch literature, referred to the "probably adverse consequences of incomplete adaptation of the human frame to the erect posture" After discussing changes in the chest and internal organs in detail, he mentions signs of insufficiency of the skeleton. These, he says, are particularly apt to appear if one of the organs is too weak in constitution or is temporarily in a weakened condition, or if social exigencies make

heavy demands on the bearing power of our locomotor apparatus. The excessive pressure liable to be imposed upon the spinal column in man on account of his erect posture tends to flatten the intervertebral discs and stretch their fibrous walls, as the result of which the adjacent cartilaginous annulus may tear.

Schaap (p. 3357) considers that "the erect posture of mankind imposes an immense burden on the spinal column. Disturbances in growth and the numerous cases of arthrosis deformans in youth demonstrate its failure to have adapted itself fully as yet to the new condition". The suggestions of other authors are in a similar trend, *viz.*, Bijl (p. 94), Cobey (p. 5), Lewin (pp. 47 and 177).

Influence of constitutional on affections of the joints

Schaap's phrase, just quoted, in itself shows that he recognises a relationship between the constitution and premature arthrosis deformans.

While suggesting criteria by which to judge whether certain disorders are or are not the result of an injury, Vogelenzang expresses the opinion that the constitution plays an important part in causing lumbago (p. 39). Although he accepts trauma as the cause of backache and lesions of the vertebral joints in special cases, he emphasizes constitutional predisposition to arthrosis deformans as one criterion.

Sleeswijk (part I) describes the climacteric in man and woman and the associated occurrence of arthrosis deformans, doing full justice to the views expressed on the matter in the literature.

Van Assen shows how cartilage may be endogenously laid down dysplastically or hypoplastically. If so, it may lead to the early appearance of arthrosis deformans. To the extent that arthritis is the precursor of arthrosis, he considers the hypersensitivity to infectious disorders of joints (arthritic diathesis) as a constitutional predisposition to arthrosis deformans.

Lubberhuizen comes to the same conclusion and refers to the relevant literature. Summarising diseases of the joints, he repeats that "constitutional factors quite certainly also play a part". With special reference to the spinal column, he probes deeper still into this matter by asking himself why it is that *all* persons whose joints are put under special pressure by their work, or for some other reason, do not present the symptoms of arthrosis deformans. The same author then speaks of "climacteric arthritis", by which name he describes the incidence of the above diseases of the joints as very commonly associated with an endogenously caused discrepancy of the hormonal balance.

Zukschwerdt (p. 102) suspects that there is a predisposition to spondylosis

as his opinion that it is not yet possible to study the old problem of "body build and psyche" by accurate methods. His enquiry into the relationship between body build and disease did not produce convincing evidence in a positive sense, contrary to the conclusions arrived at by other authors, like Draper, Parnell, Catch and Eertmark (see Brouwer).

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Verbiest (p. 689) bears witness to his belief in the constitutional influence upon back complaints by his formulation of the clinical picture of (relative) stenosis of the vertebral canal.

Kinnier Wilson (p. 1430): "Since narrowing of lumbo-sacral foramina constitutes the significant feature of the *condition* . . ." (P. 1431): "Pathogenic significance should with greater probability be attached to the low level at which the lumbar spine is sunk between the iliac bones in men as compared with women, for sciatica and other lumbo-sacral syndromes affect the former more than the latter, and the radiographic feature mentioned is undoubtedly found more often in the male".

According to Southworth, Willis (1937, p. 745) considers the influence of the constitution on low back pain to be greatly exaggerated, or not to exist at all. Nevertheless, he clearly associates low back pain with congenital lesions of the ilio-sacral joint. The title of his article (1941, p. 410) is: *Anatomic variation and roentgenographic appearance of the low back in relation to sciatic pain.*

Lewin (p. 47) mentions congenital malformations among the main aetiological factors of back disorders.

Thurel (p. 17) mentions variations of the cauda equina in association with symptoms.

Jackson devotes an article to "The association between certain anatomical facts, normal and morbid, and the symptomatology of intervertebral disc protrusions in the lumbar region".

Bradford and Spurling conclude their Introduction with the remark that "certain phenomena which have been observed repeatedly have been given acceptance although no satisfactory explanation is known" (p. 8). The discovery of "subsurgical" hernias does not always provide the desired explanation (p. 111), much is still obscure in the pathology of the lowest two intervertebral discs (p. 102). It is to be inferred from the narrative that both authors are alluding to as yet undiscovered congenital influences. Elsewhere they express themselves more clearly. Thus: "Anomalies of the lumbo-sacral spine must be suspected of causing sciatic pain" (p. 83). "Although in the individual case one must not be too quick to attribute low back and sciatic pain to an anomaly, there can be no doubt that the incidence of anomalies is greater in a series of patients complaining of low back pain and sciatic pain than in a control series" (p. 117).

Armstrong: "It should also be remembered that skeletal anomalies in the lumbar region are commonly found in association with disc lesions" (p. 114). "It is doubtful whether the majority of abnormalities have any clinical significance, although most observers have the impression that their incidence is

due to the constitution generally, but more particularly in association with anomalies of the vertebrae.

Constitutional influences upon the condition of the tissue apparatus

Van Assen (Sleeswijk, p. 544) describes general metabolic disorganisation, meaning "of a constitutional nature", and, resulting from this, a particular structure of the mesenchyme, as causes of the degeneration of joints.

Zulch speaks of "konstitutionelle Ansprechbarkeit des Bindesgewebes".

Exner (pp. 99 and 107) calls this "Bindegewebesschwache". He comes to speak of it in connection with "constitutional anomaly", under which he collects the common predisposition to osteochondrosis (lumbar and cervical), periarthritis humero-scapularis, Sudeck's atrophy, Dupuytren's contraction, flat feet and splay-feet, habitual dislocation of the patella, spondylolisthesis and numerous abnormalities of internal organs. He mentions Von Bergman's nomenclature, *viz.*, "vegetative Stigmatisation".

Zukschwerdt is another who believes that spondylolisthesis derives from a constitutional weakness of the tissues.

The constitution in relation to degeneration of the disc

Several authors consider that the widespread incidence of herniations points to a constitutional predisposition to this kind of lesion.

Magnuson associates the occurrence of hernia nuclei pulposi with the constitution.

Forestier, Zukschwerdt and many others infer the same with respect to degeneration of the disc.

Ernsting (p. 33) speaks of a "constitutionally weak back".

In his book, Lewin refers several times (pp. 54, 91, 253 and 482) in positive terms to the association between the constitution, anomalies of the spinal column, etc., and the occurrence of, or predisposition to, low back pain. In 31% of patients medically examined for jobs involving strenuous work, signs were seen of a congenital anomaly. After the elimination of these persons, as being unfit for hard work, absenteeism due to low back pain dropped by 82%. In his opinion, individuals who, under physical strain, develop low back pain and whose back becomes "locked" are constitutionally predisposed to this disability.

The relationship between constitution, congenital anomalies and back complaints

Several authors who had no doubts about this relationship have already been mentioned in the preceding pages.

CHAPTER 5

SIGNIFICANCE OF ABNORMAL MOVEMENTS

INTRODUCTION

The assumption of a certain posture is, fundamentally, the activation of part of a pattern of movement, no more no less. This being so, there can be no objection to defining both as "motoricity".

By *motoricity* we mean a complex of functions of the muscular, nervous, joint and skeletal apparatus, through the co-ordination of which parts of the body move in relation to each other (or to the outside world) or oscillate around an equilibrium, so that a posture is assumed. *Motoricity* is said to be *normal* when the individual performs this movement with the plenitude, celerity and amplitude customary to him or her when in unimpaired health.

Motoricity is *abnormal* when the movement takes place in an unusual manner for the individual concerned. "The physician and educator speaks of abnormal postures and movements, meaning, among other things, those which conflict with nature, with the purpose of the functional relations" (Buytendijk, p. 541). One person, A, can lift a weight of 100 lbs. without turning a hair, while a 50-lb. weight would put too much strain on another (B). A's powers of reaction may be so alert that they can abruptly muster a defensive mechanism without undue effort, whereas the less well-trained B under similar circumstances will strain a muscle or suffer some other lesion. A contortionist's feats are not necessarily made possible by an abnormal motoricity, since they are performed by a body pre-formed for them, either constitutionally or by training; but even the simplest of these movements would be abnormal to someone not thus conditioned, because the immense amplitude of flexion in the pattern of movement far exceeds that of the average individual

The criterion for normality or abnormality of movement need not be anchored to the condition of the whole individual, but can be provided by the condition of one of the organs taking part in the actuation of the movement. The work of the extensors and flexors should be complementary; if the necessary nervous stimulation is lacking, because the *nervous system* is diseased, the scope of the normal sequence of events constituting a movement is restricted. The same would apply if the *muscle* itself were affected. Through

higher in patients with disc lesions than in people whose lumbo-sacral spines are functionally normal" (p. 99).

We think we may conclude from this review of the literature that many authors of high repute unambiguously express the opinion that sufferers from low back pain and/or radicular pain are predisposed to the complaints by physically manifested constitutional factors.

Some other authors subscribe to this view to the extent of admitting that, although the constitutional activation has not, in their opinion, yet been proved, they consider it to be highly probable.

need not necessarily proceed from the "abnormal". The word means: "departing from the norm". Its applicability is subject, for one thing, to personal judgment, except when the norm can be established by accepted standards (*e.g.*, statistically and with corrections for dynamic changes with time).

The word "pathological" is so widely used that it would be idle to attempt tying it to a general definition.

For instance, when a catatonic schizophrenic keeps his outstretched arm raised for hours on end, he is, to begin with, exhibiting abnormal motoricity, as the time during which he assumes this attitude departs from the normal. Besides this, it is a symptom of a recognised pathological condition. *The performance of the action* is pathological, because the intention of the patient to maintain that attitude is alien to the feeling of healthy people generally. Lastly, the *action itself* is pathogenic if it should produce complaints or injurious consequences to the patient or one of his organs.

If one studies the photograph of a trained athlete' going all out" and looks at his taut muscles, one may come to the conclusion that, although the well-



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the locking of a *joint*, due, say, to a joint-mouse, a normal movement becomes an abnormal one directly its amplitude is forced beyond the limitation imposed upon it by the disorganised joint.

The work of the various organs taking part in a movement has to be *co-ordinated* to achieve a conscious or unconscious purpose, the realisation or limitation of which depends upon the constitution of the individual. Then and then only is motoricity truly normal. This is what Glorieux must have meant when he defined micro-trauma as a result of unco-ordinated movement (sudden reaction on stumbling, wricking, "wild" movements on losing hold of something, slipping off a pedal, reactions to unexpected stabs of pain, coughing in an unnatural attitude, and so forth).

Against these we have to set those movements which result from disrupted co-ordination, whether nervous or psychic; the differences between the two categories are obvious. Glorieux assumes that the muscles, joints and skeleton function normally while the nervous system and psyche satisfy the normal demands for co-ordination. A super-conditioned sportsman, *e.g.*, a light-weight boxing champion, might be capable of producing the extra mobility suddenly imposed by the situation; the external imperative might be too sudden, too exacting, for a body with "merely" normal co-ordinated functions, evoking clumsy, infelicitous, "unco-ordinated" movements. In such a case the lack of co-ordination *results from* an external impact and an injury suffered is then the result of an accident.

In the cases we have in mind, the muscles, joints and skeleton are sound, the ground of their disorganised co-ordination being primarily nervous or psychic. Clumsy, perhaps abnormal or pathological movements are consequences of the disrupted co-ordination A condition may then possibly prevail foredooming the individual to becoming the victim of a trauma and to sustaining injuries from a traumatic event If such a person trips on the pavement or elsewhere, it will be difficult to show that the injuries sustained were the result of a trauma.

Under the conditions defined, movements resulting from disorganised co-ordination will readily be qualified as abnormal (If someone with disrupted co-ordination were to perform a co-ordination test very slowly with great concentration, the time discrimination would still obtain, because the performance of the movement was abnormally slow.)

Another distinguishing feature of the uncompensated movement referred to above is that it occurs abruptly, violently and, in many cases, once only, whereas the effect of abnormal motoricity due to neurogenically and psychically disrupted co-ordination is chronic, insidious and often unnoticeable.

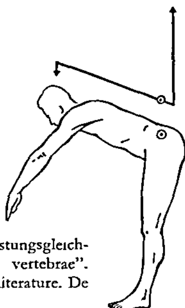
"*Abnormal*" cannot be equated with "*pathological*". For, injurious effects

Neurogenic disturbances.
Psychogenic disturbances.

Strain upon the Spinal Column in General

It is necessary to bear in mind that *strain* is always pathogenic. Those who speak of it mean the effect of an apparently normal, though heavy, burden with harmful consequences to the individual. Were one not to make this proviso, it would go without saying that pathological consequences would invariably be found when studying the influence of strain. Taken in this sense, then, it appears that, under certain circumstances, all authors consider heavy strain on the back to be pathogenic.

As has been stated before, through a certain "lever mechanism" brought into operation by lifting a weight, the forces acting upon the spinal column are multiplied fourteen to fifteen times (Armstrong, p. 32). Like other authors, he shows that, when a weight of, say, 100 lbs. is lifted in a given posture, a pressure of 1,000 to 1,500 lbs. comes to bear upon a particular part of the spinal column. It is the effect of the forces evoked by this pressure to which strain is ascribed.



The nomenclature probably derives from von Schans, who called "eine Storung des Belastungsgleichgewichtes an der Wirbelsäule": "insufficientia vertebrae". Chapchal introduced this term into the Dut-h literature. De Sèze called it "Surmenage fonctionnel".

It would be tedious if we mentioned all the authors who refer to strain. Let it suffice to mention Van Assen (Sleeswijk, p. 542) and Ernsting (p. 22) in his monograph on hernia nuclei pulposi, for the Dutch literature. In the monographs of Krayenbuhl and Zander, bearing the same title, it is pointed out that it is undoubtedly significant and relevant to the very common occurrence of herniation between the second and third decade that the greatest tension is generally imposed upon the body at this period of life.

Disturbed Motoricity

Postural changes

We now have to consider how mere physiological pressure can cause strain.

adapted motoricity to this type of work may be normal for this particular person, it would certainly be abnormal for someone who is not an athlete and, moreover, would in all probability be pathogenic as well, since sprains, stretched muscles and so forth could scarcely fail to be the result. Indeed, it remains to be seen whether the normal motoricity of an over-trained athlete or sportsman be not pathogenic since, many years later, disorders might appear, due ultimately to the preceding excessive training. This is by no means imaginary. We are not thinking primarily of the so-called "athlete's heart", arthrosis, habitual dislocations and the like; but we have noticed that people who, for whatever reason, have suddenly broken off intensive training, begin, in two or three years' time, to complain of diffuse low back pain. People who are transferred from a job involving active physical work to a supervisory one fall within the same category. Although the "traumatic" hernia certainly occurs among young people actively engaged in sport, we have found disc degeneration to be more common among athletes and sportsmen past their prime. We venture to suggest that, apart from some exceptions, pathological conditions are induced in these cases, not by the excessive physical movements themselves, but by the failure to maintain the over-trained locomotor apparatus in condition. We found, *mutatis mutandis*, the same opinion expressed in Jongbloed's book on the medical aspect of sport. The stepped-up demands on the body, kept on the stretch for many years, have to be adapted to the constitution, certainly, but so also must be the gradual relaxation. Then, though the overtaxed discs must inevitably deteriorate, they might do so by the more merciful process of fibrotic degeneration common in the elderly, rather than follow the subacute hyaline course found in the young.

With the above considerations in mind, we shall from now on call an abnormal motoricity pathogenic if it produces complaints or symptoms.

The remainder of this Chapter will be devoted to an examination of the literature for attributions of complaints to abnormal motoricity, by which it is implicitly pathogenic

Several authors, who will now be quoted, differentiate the ways in which abnormal motoricity may be brought about We shall therefore subdivide the matter as follows·

A. Strain upon the spinal column in general.
B The effect of disturbed motoricity associated with.
   Changes in spinal posture
   Lesions of the vertebral joints
   Degeneration of the disc
   Other disorders of the vertebrae
   Abnormalities in the extremities

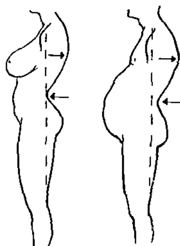
of the spine (Schmorl, p. 235), is capable of resulting in lumbo-sacral postural changes. On radiographs of patients complaining of low back pain, it is not very rare to find old impression fractures at a higher level, where the pain is *not* localised. In such cases the chronic strain due to the lumbo-sacral postural changes has resulted in a disc lesion at a lower level, or in another painful mechanism (functional listhesis).

Edinger (Congress on Chiropractice, Freudenstadt 1956) put the foregoing in a nutshell, thus: Postural changes have to be absorbed by muscular strength; such a compensating mechanism may be lost through an illness; the resulting decompensation brings extra pressure to bear on the skeleton.

Martius (quoted by Ruding) writes that the greater the muscular compensation needed for defective statics, the sooner is the limit of pressure tolerance exceeded. On the analogy of symptoms produced by flat feet, he suggests that, through changed statics, certain groups of muscles have to bear extra weight and "fatigue pain" is the result.

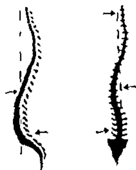
Fincher (quoted by Lewin, p. 739, when discussing race as an aetiological factor in backache) finds that low back instability in the white is about four times more common than in the coloured (negroes), but the ruptured disc proportions are about equal.

The following among many other authors affirm a relationship between low back pain and/or radicular pain, on the one hand, and the assumption of a pathological posture and the performance of pathological movements, on the other: Bradford and Spurling (p. 119), Boeters (Erbbiologie handbook), Brown (J. Bone and Joint Surg.), Buytendijk, Friberg and De Sèze (3rd Rheumatism Congress, Scheveningen), Goinard (p. 154), Junghanns, Kendall (1952), Lewin (pp. 51, 53, 480), Matthiash (Brocher, p. 1), Mol, Schaap (p. 35), Vogelensang, Zukschwerdt (p 69).



Here are one or two striking facts which some of them recount: Van Assen describes how low back pain may be produced by a pendulous abdomen. The posture assumed to compensate for the weight puts a strain on the back. Swag-bellied patients come to consult their medical adviser about lumbar pain. This bears out a case of our own. The patient complained of low back pain; her breasts were so hypertrophic that her posture had changed to compensate for the extra weight that had to

Let us not lose sight of the fact that the skeleton functions as an integrated whole. For instance, Th.XII and L.V are geared together (Cramer). When, for whatever reason, L.V is pushed forward, Th.XII should automatically shift backward. "Vorneigung L.V = Rückneigung Th.XII". The spinal column is then said to be compensated. If this compensating mechanism in the attitude of the spinal column is prevented from coming into operation, other mechanisms are called upon to do the job, as, for example, pelvis displacement forward, or a thoracic kyphosis. Cramer demonstrated this in extensive material. He has it that these extra compensating mechanisms strain the spinal column and produce symptoms.



Sollman observed identical compensating mechanisms after dislocation of the atlanto-occipital joint. The resulting local obliquity of posture was absorbed by curvatures of the spinal column elsewhere, distal to the lesion. This shows that the cause of a postural abnormality of the lumbo-sacral spine may reside in the cervical spinal column.

Guttmann came to the same conclusion, remarking that, within 3 years of beginning to wear a certain kind of wimple, a great many of the nuns he examined had a curiously stretched cervical spine, which he called a "swan's neck"; to compensate for this, the rest of the spine was kept fixed in a different position. De Sèze also decided that scoliosis is often induced by postural changes elsewhere. Zukschwerdt suggests that curvatures of the spine are for the most part caused by internal diseases, such as of the bladder and kidneys

Van Nes accounted for scoliosis, homolateral or contralateral, as a symptom associated with hernia nuclei pulposi; and the functional X-ray examination of the spine in various positions, as practised by Hadly and Hassin (recorded in the Dutch literature by Weersma), provides a useful means of distinguishing between these entirely different postural changes. The intervertebral disc remains mobile in the case of scoliosis resulting from distal pathological conditions, but it is held stiffly and produces local pain in the case of scoliosis resulting from a lesion in the disc itself. How this is with hereditary scoliosis, of which Schmorl writes, we do not know; it is, however, a remarkable fact that, according to Van Assen (Sleeswijk p. 553), people born with a crooked spine "never" complain about backache.

By now it must be clear that a local abnormality of the spine is liable to produce postural changes elsewhere in the spinal column. Thus inflammation, or perhaps the residual condition of a trauma affecting the thoraco-lumbar part

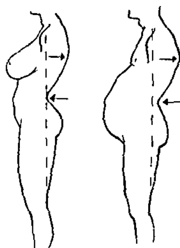
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Here are one or two striking facts which some of them recount. Van Assen describes how low back pain may be produced by a pendulous abdomen. The posture assumed to compensate for the weight puts a strain on the back. Swag-bellied patients come to consult their medical adviser about lumbar pain. This bears out a case of our own The patient complained of low back pain; her breasts were so hypertrophic that her posture had changed to compensate for the extra weight that had to

be carried. Possibly the inferiority complex which the imposition of this inaesthetic burden had set up helped to disorganise her psyche, but it was obvious that the patient was suffering not only from the mental but also from the chronic physical strain of this abnormality.

Loujot and Bélart (3rd Rheumatism Congress, Scheveningen) found that, depending on the pressure, spondylotic changes are liable to take place in various parts of the spinal column. If, for instance, a workman always carries heavy loads on his right shoulder, the symptoms are presented predominantly in the left half of the body. Generally, the changes in wrestlers and shot-putters are ventral in the vertebral bodies.

Lesions of the joints

Schmorl says the pathologist is able to show, anatomically, the fracturing of the end-plates, followed by ingrowth of tissue at the site after strain. Degeneration of the disc takes place secondarily (p. 133).

Van Assen (Sleeswijk, p. 547) observed synovial rents due to excessive movements.

Lubberhuizen (Sleeswijk, p. 626) comments on the fact that strain due to static as well as functional factors leads to osteoarthrosis in some and to *chondral* wear and tear in others.

Kuhlendahl seems to think that the harm done by the sudden impact of mechanical factors is due to the fact that the *elasticity* of the tissues has no time to come into action; consequently, disturbed motoricity, in the broadest sense of the word, causes a lesion of tissue, owing to which there is a secondary reaction of the *bone*, with spondylosis and spondylarthrosis as the result.

The part played by disturbed motoricity in producing spondylarthrosis

As we found in a preceding chapter, it is generally agreed that spondylarthrosis is liable to produce pain.

We do not yet know exactly how this pain is induced. We would recall that spondylarthrosis can be detected radiographically, yet the patient experiences no pain. Alternatively, the patient's case history and, possibly, examination may show the typical syndrome of spondylarthrosis, yet no abnormalities show up in the radiographs.

It is difficult to explain why the pain disappears after deep X-ray treatment of an arthrotic joint, though the same abnormalities subsequently appear in the radiographs. During the Third Rheumatism Congress (Scheveningen 1955) the results were given of X-ray therapy applied to patients with arthrotic joints. The patients with identical changes in the joints were divided into two groups, one of which received X-ray treatment, the other being "X-rayed" by a machine that was not working. The results in the members of both groups were the same.

[illegible] the complaints [illegible] [illegible] from a bilateral arthrosis deformans of the hip joints, owing to which she could barely move. After Impletol injections in the retro-ovarian cavities, the pain ceased instantaneously and the patient was able, despite her destroyed joints, to walk; yet radiographs – also made a considerable time afterwards – showed that the abnormalities of the joints had not changed at all.

It is also known that a painful arthrosis deformans induces postural anomalies and disrupted motoricity.

What we should now like to know is whether the reverse is true, viz., whether disrupted motoricity causes changes in the joints, notably spondylarthrosis. Perhaps we can find the answer in data recorded in the literature. Schmorl (p. 245) and Van Assen (Sleeswijk, p. 551) describe a fibrotic change with wasting of the joint capsule and supporting ligaments as the result of insufficient activity of a joint. On the other hand, Schmorl (p. 242) points out that spondylarthrotic changes are associated with the mobility of the parts of the skeleton involved.: "Am häufigsten und stärksten sind aber meist die beweglichsten Teile der Wirbelsäule (Hals und Lendenteil) von Arthrosis der kleinen Gelenken befallen". "Es muss hier ausdrücklich erwähnt werden, dass an allen Stellen, an denen Wirbelkörper unbeweglich mit einander verwachsen sind keine Randsacken entstehen" (p. 209). He goes on to say that post-traumatic spondylarthrosis is not presented until the patient has become mobile and that the spondylarthrotic hooks — if any – disappear again after the patient has had a period of bed rest. He thinks the investigation pursued by Beneke and Simmonds (p. 207) provides indirect evidence of the association between disrupted motoricity and the incidence of spondylarthrosis. It had already been observed that spondylarthrosis is more common on the right of the spinal column, which was accounted for by the right-handed habit of the individual. To verify this, viz., that right-handedness brings more pressure to bear on the right-hand part of the body, an enquiry was made into the position as it is in left-handed people. Schmorl (p. 207): "Und tatsachlich wurde bei Linkshandern starkere Randwulstbildungen an der linken Seite der Brustwirbelsaule gefunden".

Armstrong (p. 146) states that if the disturbed motoricity persists for many years, degenerative changes take place in the intervertebral joints.

Zukschwerdt (p. 174) and Van Assen (Sleeswijk, p. 551) attribute the occurrence of spondylarthrosis to painful muscle tensions which have disturbed motoricity.

Van Assen (Sleeswijk, p. 548) has pointed out that a disturbance in one

joint may produce (spondyl)arthrosis in another, an opinion which we have found several times in Schmorl's work as well. If, say, the knee joint has undergone arthrotic changes and arthrosis of the hip joint also occurs owing to the resulting disturbed motoricity, no difference at all is to be found, either at X-ray or pathological examination, between these two joints, yet one may and the other may not produce symptoms.

Finally, disturbed motoricity involves changes not only of the joints but also of the intervertebral foramina. Schmorl (p. 24), and especially Zukschwerdt (p. 991), who devotes a chapter to it, as well as many other authors, have established this change at autopsy, noticing at the same time that the root involved was compressed through these changes.

Indeed, we may conclude, in company with Schmorl (p. 207), that disturbed motoricity leads to spondylarthrosis of the spinal column.

Disturbed motoricity and the disc

It is known that a disc lesion can produce pain and that a painful disc is liable to produce anomalies of posture and movement.

Osmond-Clarke (Armstrong, p. VI): "It is very rightly pointed out that a degenerated disc lesion produces a mechanical disturbance of the spine . . .".

Lewin (p. 253): "Degeneration of a disc leads to instability of at least one vertebral body".

Again we ask ourselves whether, conversely, disturbed motoricity leads to a disc lesion. The answer is, actually, supplied indirectly by the point made that disturbed motoricity causes spondylarthrosis and it is known that spondylarthrosis may lead secondarily to degeneration of the disc (Van Assen, Sleeswijk, p. 547). We can, however, find a direct answer in the literature.

Armstrong (p. 34) suggests that disturbed motoricity (as the result of the assumption of the upright position) leads to disc degeneration.

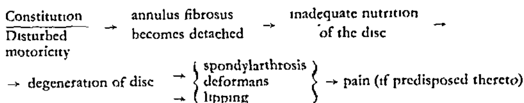
Thiebault thinks it is even possible for a disc prolapse to take place secondarily as the result of sciatica accompanied by postural changes of the spinal column involving unequal pressure on the discs.

Schuller accounts for disc lesions by assuming that inflammation of muscles or nerves, as for example in rheumatism, induces postural changes which produce spondylosis and, secondarily, lead to disc degeneration.

Schmorl (p. 133) visualizes slight strain as producing fissures around the disc due to secondary fibrosis, with resulting degeneration of the disc. On page 177 he expresses the opinion that a secondary intervertebral chondrosis is caused by unbalanced pressure upon the intervertebral disc involved, in association with the most divergent causes of disease.

The foregoing implies a vicious circle:

If the subject is constitutionally predisposed that way, his motoricity becomes disturbed, owing to which minor traumatic lesions occur in the vicinity of the intervertebral disc; this receives insufficient nutrition; spondylarthrosis develops and the disc degenerates, producing pain; in its turn, this pain may foster or activate motoricity disturbances.



Disturbed motoricity and disorders of the vertebral body

Schmorl (p. 164) describes how all parts of the spinal column, especially those forming a "mobile segment", are so delicately attuned to each other "dass jede Storung eines Einzelteiles die anderen in Mitleidenschaft zieht". This agrees with our own view that the spine is to be regarded as a single functional unit, so much so, in fact, that disturbances in the upper part of the cervical spine, for instance, may be pathogenic to the lumbo-sacral part. Accordingly, it is necessary to realise that, as Schmorl writes (p. 107), the effect of a trauma is not confined to any one particular site in the spine, where it remains clearly visible radiographically, for instance, but ramifies to several other parts, where, through muscle tensions, small ruptures and dislocations may occur. This kind of post-traumatic changes may be expected to attend all conditions involving tonic or clonic spasms (epilepsy, electric shock treatment and so forth). We had ourselves noticed that several patients suffering from hernia nuclei pulposi in the usual L.IV – L.V or L.V – S.I localisation had the record of a previous trauma, the manifestations of which at a higher level were radiographically visible. We had already inferred that the residual conditions of the trauma had led to unbalanced motoricity and that, as a result of these abnormal movements, too much pressure had come to bear upon the lower lumbo-sacral segments, causing a disc lesion secondarily with herniation. We found support for this view in the observations reported by Brocher (p. 50) and Descuns (p 385) to the effect that nuclear retropulsion of the L.IV – L.V or L.V – S.I disc had occurred a considerable time after a previous trauma elsewhere in the spinal column. "Nous voudrions cependant attirer votre attention sur la répercussion discale, à distance des vertèbres lésées au cours de certains traumatismes".

Lastly, the same mechanism which comes into operation through fatigue, muscular pain and minor distortions elsewhere in the body, must be held responsible for low back pain. San Giorgi and Frijda look upon developmental defects, such as transitional vertebrae, morphological anomalies and the like, as the cause of discordant movements which, in turn, produce symptoms. Ruding believes that transitional vertebrae and spondylolisthesis bring about a disc lesion by the same mechanism (*via* postural anomalies, strain and muscular tension).

Disturbed motoricity due to abnormalities of the extremities

There is a good deal of truth in the popular notion that many back complaints stem from trouble in the extremities. It is emphatically stated in the literature that abnormalities of one joint in an extremity can lead to abnormalities and symptoms in another joint, either in the same extremity or in a contralateral one. Van Assen (p. 633) reports the case of a patient with clearly defined arthrosis of one hip; later the check radiographs showed that the same changes had taken place in the joints of the other hip. Cases are also known of patients complaining of pain in the knee, whereas radiographically the causal abnormality is found to be in the hip.

We now propose to show how far the literature goes to adduce evidence of morphologically demonstrable changes in the lumbar spine as the result of peripherally disturbed motoricity; also how chronically disturbed motoricity of the extremities produces low back pain and, possibly, radicular pain.

First let us quote Finneson and co-workers, who as late as 1957, wrote: "The question whether amputation with prosthetic devices and the resultant changes in mechanical stresses on the lumbar spine hasten the onset of discogenic symptoms cannot be answered on the basis of available data".

Against this we have the following pronouncements:

Van Assen (Sleeswijk, p. 547) shows that residual curvatures after a fracture of the lower extremities may later lead to spinal complaints. In the spinal column, too, the cartilage of the intervertebral discs degenerates in the places under most stress.

Bradford and Spurling (p. 112) hint that many patients complaining of low back pain have been suffering for some considerable time from orthopaedic abnormalities in the lower extremities

Lewin (pp 46, 52, 175 and 640) says unequivocally that paralysis, underdevelopment or disorganised movement of the muscles, shortening of one of the legs, etc. are by no means seldom the cause of back trouble. "A fixed hip or an uncorrected short leg throws an increased mechanical strain on the

lumbar spine". "The continued use of one's back in a poor mechanical posture produces pain". "There is often a reciprocal between back and foot, ankle, knee. It is a mechanical chain reaction". He says all this while discussing patients complaining of constant fatigue and a gnawing pain in the lumbar spine. In his view, the reason why a short leg upsets the balance of motoricity is that the back recruits compensatory mechanisms to correct the consequences of the peripheral malformation (scoliosis, circumduction, etc., etc.).

Edinger (Chiropractice Congress, Freudenstadt, 1956) shows that this form of motoricity is associated with the pelvic tilt and that the latter can be called into action in countless ways. In practice, therefore, we have to differentiate between:

1. Shortening of one leg.
2. Asymmetry of the trochanter angle as the result of an anatomically demonstrable abnormality.
3. Anatomical symmetry of the trochanter angles, one of which, however, functionally assumes a different position, due in part to circumduction, abduction, adduction, *e.g.*, in association with a cerebral lesion.
4. Asymmetry of the ilio-sacral joints.
5. Pelvic tilt to compensate for an abnormality of the higher part of the spine (obliquity of head and neck → produces scoliosis → pelvic tilt).
6. Local distortion resulting from a disorder, such as arthritis and arthrosis, or owing to referred pain (reflexogenic compensatory posture for pain in an organ).
7. C-scoliosis, so called (*e.g.*, due to a wedge vertebra, but also from no known cause, often with compensation taking the form of a crooked sacrum).

Several authors have evinced special interest in statics and disturbed motoricity observed in patients who have had a leg amputated. These cases offer rewarding material for study, because the patients are as a rule in good health and the disorder is chronic. Moreover, thanks to revalidation and artificial limbs, the patients are in good enough fettle to move about a good deal; if the expected phenomena occur anywhere, it will certainly be in them.

Zukschwerdt (p. 159) holds that, as the result of disturbed statics and motoricity, say following the shortening of one leg, spondylarthrosis occurs and cartilage degenerates, owing to which the intervertebral foramina narrow and their contents are damaged.

Roenick (Chiropractice Congress, Freudenstadt, 1956) reports on an examination of 47 patients, all of whom had had one leg amputated and had had an artificial leg for a long time. To function properly, these artificial legs have to be on an average 2 cm shorter than the normal one. In the observed cases this produced a pelvic tilt with varying left and right scoliosis of the lumbar spine. He found that this had a permanent effect upon the spinal column. In

the sitting posture this pelvic abnormality disappeared; nevertheless, as he was able to verify radiographically, the posture of the whole spine had changed permanently, as was clearly proved by the fact that the dens epistrophei was held unsymmetrically in 46 cases and the spinal process of C.I abnormally in 35 cases.

Breslau (p. 43) made an extensive study of patients who had undergone amputations. Like other authors, he notes an effect upon the skeleton resulting in postural anomalies and signs of strain. What is more important is his emphasis of the fact that the resultants, notably the processes of wear and tear, depend upon constitutional factors. This undoubtedly is the obvious answer to the question why the disturbed motoricity in one patient after an amputation is pathogenic and not in another. Breslau is especially clear in his exposition of the causal relation between amputations and diseases of the vascular system. In this he makes a stand against the assumption of a causal relationship, as Leriche puts it: "les douleurs des amputés". In the latter's book, *La Chirurgie de la Douleur* (pp. 222, 224, 254), no syndromes are described in this connection which suggest low back pain and radicular pain. We have to picture the vascular syndromes – often combined with heart crises, angina pectoris, phantom pain, sympathalgia, etc. – as a complication apart which occasionally develops in cases of amputation. The facts, too, that such strong psychogenic factors play a part in producing the symptoms mentioned above and that sometimes treatment only imitative of neural therapy availed to clear up those symptoms, led us to conclude that the phenomena he had observed were of an entirely different nature from those forming the subject of this chapter.

Kostíc (p. 74) mentions extensive research into the cause of hernia nuclei pulposi among Jugoslav railway officials. The study contains many statistics, which the writer uses to prove that many people suffer from low back pain or disc lesions as the result of injury. The same statistics could be used to show that there exists a clear relation between the effect of motoricity and the symptoms

The occupations of the 500 persons with hernias verified at operation proved to be vastly different one from the other, *viz.*,

Arduous physical work	37.4%
Intellectual, administrative jobs, etc.	29.2%
Artists and skilled workers	21.0%
Household work	9.4%
Professional sportsmen	3.0%

Surprising as the high percentage of the second group may be, it tallies with

our view that persons so engaged are seldom in physical training, so that, when called upon now and again to exert themselves suddenly and strenuously, they are unable, physically, to stand up to the unusual strain.

It certainly does not surprise us that the percentage of professional sportsmen is so small in this list. It bears out our own experience. First of all, they owe their very selection to their constitution; if it were not first class, they would not become professionals. Then, and this is most important, they remain in good condition. Therefore, both their build and their motoricity enable them, time and again, to cope with a strain which might well be injurious to other constitutions.

The effect of movements is revealed still more clearly by the following figures. Of the 500 hernias verified at operation:

67 % occurred in persons whose occupation necessitated standing;
33 % occurred in persons engaged in sedentary work.

Cobey devotes a monograph to postural back pain. On page 18, for instance, he describes the causes of a poor posture, *viz.*, weak abdominal muscles, pendulous abdomen, pregnancy, residual conditions of infantile paralysis, leg fractures, leg amputations and pelvic tilts. Owing to this bad posture (and the associated pathological motoricity), stress is imposed on the lumbo-sacral joints and ligaments and this causes spondylarthrosis. If the subject is predisposed, this stress causes back pain.

Low back pain occurs in 38 % of housewives,
in 35 % of office workers (week-end athletes)
and in 2 % of workmen. (Hauser, quoted by Cobey, p. 35).

He attributes this to the bad posture assumed in the various occupations, describing it admirably thus: "The average housewife, who must do heavy work in many positions, does not protect her back by proper development and use of muscles. In general, her muscles are neither strong nor well balanced. Her posture is usually very poor, with sway-back and protruding buttocks, increasing the lumbo-sacral angle and thus the shearing force upon the joint. She is often fat, with a tendency to a pendulous abdomen. The ligaments tend to become inelastic, thin and tear easily. To add to her difficulties, she teeters around on high heels, balancing herself by a forward roll of the pelvis. In this position she stoops instead of squats to pick something off the floor. With the forward roll of the pelvis, partial flexion of the hips is already present. They will flex only so far, then something must give. The contracted lumbosacral fascia and its joints must give. The capsules of the joints, or the surrounding

fascia, are torn". "The office worker develops similar difficulties in a different way. By sitting continually at a desk, he develops short hamstring muscles, contracted muscles of the gastrocnemius group in the leg and of the hip flexors. He rides forward on the pelvis and lets it roll under him, increasing his lumbosacral angle. His paravertebral lumbosacral fascia and muscles become contracted and when he attempts to stand, his low back is still in the attitude of sitting. With the distortion of posture, his week-end attempts at athletics or house- and garden chores put an unusual strain on the lumbosacral joint!"

Then follow some examples, such as the attitudes of the dentist and shop-girl, and finally: "All these groups may be contrasted to the laborer who has learned how to use his muscles and is accustomed to using them".

An analysis of 1154 cases of low back pain (examination of American Army) disclosed that its causes were as shown in the following statistics (Cobey, p. 36):

60 % as the result of postural changes.
10 % as the result of spondylolisthesis.
10 % as the result of spondylarthritis.
5 % as the result of spondylarthrosis.
5 % as the result of hernia nuclei pulposi.
10 % from psychogenic causes.

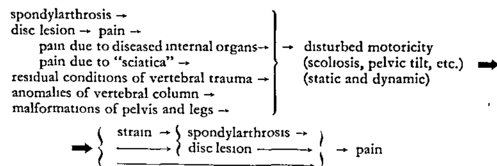
Cobey (p. 35) points out that low back pain is very often due to an unusual placing of the feet, mentioning the study made by Hiss, who asserted that 40% of sufferers of backache as the result of postural abnormalities also complained of painful feet.

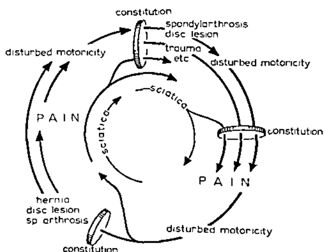
Cobey (p. 164) advises us, when applying conservative treatment to improve posture, to pay due attention to the whole statics of the spinal column and the extremities Much as he approves of physiotherapy for the prevention of low back pain, he comes to the conclusion that at a given moment permanent changes will have taken place in the spine, against which physiotherapy cannot prevail (pp 43 and 44). Pathological movements cause anatomical changes – demonstrable by the pathologist – in the muscles, spinal column and intervertebral discs which are permanent and unaffected by physical exercises. According to him, chronically disturbed motoricity causes narrowing of the intervertebral foramina If there is simultaneous oedema of the roots, a radicular syndrome may be produced, so that a poor posture may even bring on sciatica. At this stage his advice is to cease bothering the patient with conservative treatment and considers facetectomy (i.e. foraminotomy or decompression of the intervertebral canal in some other way) to have become imperative.

Let us conclude by quoting the greatest authority on this matter, meaning, of course, Schmorl (p. 216):

"Eine dauernde Überlastung als Ursache fur eine Spondylosis deformans ist bei Störungen des Beckens und der Beine möglich: Muskellähmungen, Beinmissbildungen, Beinverkurzungen, schiefgeheilte Beckenbrüche, Beinamputationen mit und ohne Prothese". He speaks of "Skolioseformen verursacht, durch ausserhalb der Wirbelsäule selbst liegende Erkränkungen und auf Schmerzreflexen zuruckzuführende Skoliosen" (p. 233). He concludes his textbook (p. 254) with the words: "Änderungen im Bereich der unteren Gliedmassen (Beinverkürzungen, Plattfüsse, Gelenkversteifungen u.s.w.) bedingen unter Umständen statische Kreuzschmerzen".

In the physical sphere – disregarding, for the moment, the possible consequences of neurogenically or psychogenically disturbed motoricity – the vicious circle can now be closed as follows:





This diagram is intended to demonstrate that the transition from one phase to the other depends on the existing constitution (possibly on acquired characters, *i.e.*, the condition).

The data cited in the foregoing from the literature substantiate our view that disturbed motoricity is capable of producing back complaints. The reason why it does in one case and not in another similar one is, we believe, because one constitution is predisposed to such complaints and the other is not. It would seem that precipitating factors also have some quantitative influence: should a movement be so gravely hampered that it barely takes place at all, should the disturbance have arisen recently, or should someone, say, with one leg be confined to bed for other reasons, then the effect of the disturbed motoricity would scarcely be noticeable.

If, after making the above allowances, the reader is able to accept the postulated influence of disturbed motoricity, he will also be willing to follow the next step in our reasoning. Surely the *cause* of the disturbed motoricity is immaterial? Once it has been inferred from convincing evidence that a given disruption of movement is pathogenic by virtue of its intensity, frequency and duration, it follows, *ipso facto*, that every disturbance satisfying the same conditions is liable to involve the same pathological consequences.

Most commonly, motoricity is disturbed neurogenically, as will be obvious, if, that is, we leave the finer shades of a mildly disturbed psycho-motoricity out of consideration We can demonstrate by our own material that a neurogenic disruption of motoricity in the locomotor apparatus is capable of producing low back pain and radicular pain. We have not been able to adduce significant proof of this from the examination of a large number of patients; in fact, it does not seem to us to be feasible in view of the many factors that have to be taken into account.

There is little hope of finding an answer in the literature to the question whether disturbed motoricity due to neurogenic disorders is known to be capable of producing these complaints, because, as far as we are aware, this question was never asked in the past. Little value can be attached to an anamnesis, and less still to an abstract of it made for case histories published in the literature, if the examiner has not been on the look-out for a certain complaint or phenomenon It is our experience that a patient is apt to ignore secondary complaints when he is engrossed in the gravity of his primary illness. Furthermore, many neurogenic disorders would have to be eliminated from a study of the kind intended to satisfy the conditions set forth on page 20;

for, the disease will have to have existed long enough to allow secondary changes and complaints to be presented. Nor should the gravity of the disease be such as virtually to inhibit the exercise of an already disturbed motoricity. The disease would also disqualify if it were itself associated with aches and pains which could be mistaken for radicular or back pain, though the morphological manifestation of secondary changes due to the disturbed motoricity could be allowed to be a clear indication. The neurogenic disorder must not itself bring about morphological changes (*e.g.* trophic disturbances) in the innervation areas of the lumbo-sacral roots; in that case even the occurrence of a typical low back syndrome cannot be accepted as an indication. For these reasons we have to disregard the anomalies of the cauda and the dura, as also myelodysplasia, perfectly as they otherwise fit into the framework of this subject.

Within these strict limits and confining our quest to a few textbooks and monographs, we set about searching the literature for an answer to this question, little expecting to find it. Yet, surprisingly enough, we found many references which suggest that a careful study of extensive patient material would produce an answer in the affirmative.

Disorganised motoricity due to neural disturbances

As was stated in Part I, we have noted the occurrence of low back pain and/or radicular pain associated with *residual conditions after lesions of the nervous system,* operations for hernia, trauma capitis or an affection of the spinal cord or peripheral nerve. Some examples are known of the following cases, *i.e.*, meningioma, pontine angle tumour, glioma (!), intracerebral haemorrhage, hernia nuclei pulposi cervicalis. The symptoms occurred after one or two years in patients who remained mobile in spite of body-plan disturbances (paresis, ataxia). We have not found similar reports in the literature. We found that this is to be explained by the fact that, upon subsequent examination, neither the patient nor the clinician associates these usually vague complaints with the primary illness, which dominates the picture.

The influence of *paralysis* of the lower extremities upon the statics of the spine has been referred to in the preceding pages (Armstrong, Epstein, pp. 122–125, Schmorl, etc.). Lewin (p. 640) says spastic paralysis generally is one of the causes of low back pain.

The *neurarthropathies* (*e.g.*, as in tabes and syringomyelia) do not come within this context. They are, however, excellent examples of the consequences of neurogenic anomalies of movement (Epstein, p. 345, Lewin, p 653, Schmorl, p. 180). The disease itself, however, induces trophic disturbances and deadens

the sensation of pain. Hence distal consequences are not necessarily inherent in disturbed motoricity. Thus Kinnier Wilson (p. 1399) says that kyphoscoliosis in association with syringomyelia is due to trophic disturbances. This non-congenital postural anomaly might conceivably be due to disturbed motoricity as the result, say, of muscle weakness, though Kinnier Wilson considers that this is seldom present.

We had the clinical records of 61 patients for an investigation into the relation between syringomyelia and low back pain. All these patients, without exception, were examined thoroughly. Eight complained of low back pain. Here, in brief, are a few relevant points:

1. Pat. 10342 complained of sensory disturbances and paresis of one arm during the past six months, in the last two of which was added back pain radiating to both legs. The Lasègue was positive on both sides; no Achilles tendon reflex.
2. Pat. 17769 suffered for the past 7 years from syringomyelia (paresis and sensory disturbances of both arms and legs when examined). Since girlhood (estimated time 30 years) had complained of low back pain. Lasègue positive on both sides.
3. Pat. 16915 had presented symptoms in the past 6 years that might be ascribed to syringomyelia. Disturbances found in the area of the cerebral nerves, disturbance in co-ordination, paresis and sensory loss in both arms. Low back pain (not stated for how long) without radiation. Lasègue negative on both sides. X-ray examination of the spine showed marked narrowing of the L.IV – L.V disc, arthrosis and sacralisation of L.V.
4. Pat. 14634 suffering from syringobulbia for past 5 years. Only disturbances in the region of the cerebral nerves Low back pain as well in the last 3 years, radiating to the right leg. Lasègue doubtfully positive on both sides. Coughing, sneezing and straining doubtfully positive. Myelographic evidence of backward displacement of L.V – S.I.
5. Pat. 23098. Sensory disturbances and paresis of one arm and one leg for past 13 years, during which period recurrent low back pain. Lasègue negative.
6. Pat 23405 suffering from syringomyelia for past 5 years with sensory disturbances in trunk and leg, paresis of one leg Lasègue negative. Had always complained of low back pain.
7. Pat 18136 had low back pain for past 3 years, exacerbated by coughing, sneezing and straining Sensory disturbances and disturbances in co-ordination, also paresis of one leg, in the last year Lasègue positive. No changes to be seen on radiographs
8. Pat. 21991 Paresis in past 5 years of one leg and non-radicular sensory loss. Low back pain in the same period, radiating to the same leg Otherwise the complete picture of hernia nuclei pulposi No X-ray examination.

In two patients the changes in the spine were enough to account for the backache; in the others there was reason to associate the low back pain with the syringomyelia causally. It should be noted, however, that in view of the small volume of the material, a significant correlation can neither be asserted nor denied

In point of fact, we know of several patients who underwent a *negative* exploration for a disc lesion on account of low back pain, with or without radicular symptoms, and, after an interval of varying duration, developed a disease of the spinal cord (dissociated sensory disturbances, muscular atrophy, diminished tendon reflexes

in the legs with pathological sole reflexes). Similar cases are known after scalenotomy for brachialgia.

Myelodysplasia (including Fuchs' syndrome, club-feet, residual conditions of meningo-myelocele) must also by excluded, because it may be associated with malformation of the spinal cord, the cauda and the lumbo-sacral vertebral column and in this way produce low back pain and also radicular symptoms.

Dystrophy of the muscles causes serious disturbances of movement, gradual at first but progressively worsening. Though he speaks of deformities (p. 975), Kinnier Wilson does not mention accompanying pain, but we suspect that this is due to a misunderstanding. We had an opportunity of observing one or two cases; they were sent to us because of back complaints. A boy, who worked himself up from the supine position by grasping his legs in the familiar manner, had complained at the early age of 9 about a weak and painful back. For an outstanding illustration of our contention that some authors failed to describe secondary symptoms (*e.g.* pain) if they did not consider them to be pathognomonic, we refer to Kinnier Wilson's chapter (p. 832) on dystonia muscolorum deformans. One has only to look at the plates to realise that these patients *must* have suffered pain as the result of their deformed attitudes, yet not a single reference is made to it in the text.

Epstein (p. 122) underlines these two maladies as causing a pathological posture. B. Brouwer (Addendum, p. 283) calls Oppenheim's dystonia musculorum progressiva (or Ziehen's chronic rotation spasm) "above all a disturbance of motility". He cites the case of Wimmer and Neel, which was accompanied by "enormous secondary movements" and which started with *diffuse back pain*. Brouwer saw the same deforming movements associated with progressive lenticular degeneration and pseudo-sclerosis, and he "could not help thinking that these maladies are variants of each other".

Kinnier Wilson (p. 806), in his comprehensive work on *hepatolenticular degeneration* (Wilson's disease), defines the associated motility disturbances as spasm with involuntary movements. The disease is painful in the acute phase (muscular spasm), but the pain disappears when the chronic stage is reached (p. 815).

According to Kinnier Wilson (p. 608), Sydenham's *chorea* is accompanied by "rheumatism", or complications of it, in 28.6% of cases. Several authors (including Prick) are of the decided opinion that Sydenham's chorea is a manifestation of rheumatism. Wilson raises the point (p. 610) as to whether polyarthritis rheumatica acuta really does play a sufficiently important part in the aetiology of chorea to warrant the term "cerebral rheumatism". Noting how many of 3546 patients suffered from "rheumatism" (affection of the joints,

growing pains, pain in joints or limbs), he found that they amounted to 75%. In 20%, signs of rheumatism did not appear until six years after the onset of chorea.

One might ask oneself tentatively what percentage of the rheumatic pains represented *pain caused by movements.*

B. Brouwer and Wilson hold the same views on that motility disease *par excellence: paralysis agitans.* Brouwer (Addendum, p. 70): "In the first period, pain is produced through the extremities. Sometimes the pain is so severe that this stage might aptly be termed a 'forme douleureuse'. It is a striking fact that the worst pain occurs in the extremity which subsequently begins to shake". Kinnier Wilson (p. 791): "Initial symptoms – pains, . . ., aching . . .–, are the counterpart of early stiffness and limitation of willed movement. (P. 798): "Pains, aches . . . etc. can be ascribed in part at least to muscular rigidity". (In this context it is well to remember that the age of the majority of Parkinson patients is the "normal" one for rheumatism, arthrosis and the like.)

We had also expected to find references to pain due to motoricity disrupted by ataxia, but not one of the authors mentions pain or postural changes.

The degenerative failing of the cerebellum, in all its variety, causes an uncertain gait, but the patients move slowly and carefully. Brouwer (p. 201) does, however, quote the description by Déjérine and Thomas of the clinical picture of olivoponto-cerebellar atrophy. These patients began by complaining of *fatigue* in the legs from no demonstrable cause.

Perhaps *Friedreich's tabes* is an intermediate case; it is an hereditary spino-cerebellar degenerative disease, marked chiefly by locomotor jerkiness and ataxia; hence its name: hereditary spinal ataxia (Kinnier Wilson, p. 943). Buytendijk (p. 455) ascribes this ataxia, with the posterior cords quite intact in Friedreich's tabes, to purely functional disturbances of sensation. As far as we can make out from his account, these set up a change in the body (*e.g.*, p. 482) which leads to disturbed motoricity. Now, Kinnier Wilson (p. 946) states that the awkward gait in the early stage of Friedreich's ataxia is accompanied by painful spasms in the legs, pain in the legs, pains such as those associated with tabes dorsalis and radicular pain. Kyphoscoliosis with lumbar compensation comes later. Apparently the patient reacts badly to bed rest; that is to say, the symptoms clear up temporarily, but worsen when the patient is again mobilised. To our mind this shows clearly how much the pain depends on the disturbed motoricity associated with Friedreich's ataxia. Brouwer (p. 214) only reports curvatures of the spine (kyphoscoliosis), without any reference to pain. In his Addendum (p. 48), however, he cites Greenfield's

description of some subacute variants which are distinguished from the classical picture by the occurrence of pain in the legs.

Holding, as we do, that the consequences of disturbed motoricity are presented in the initial phase, when the severity of the disturbance is not yet such as to impede movement altogether, it is plausible that the symptoms should wane in the chronic phase, when the patient no longer moves, and come on again when the patient can once more be mobilised. In Greenfield's cases the mobility disturbance was subacute and might therefore have induced more violent reactions.

Funicular myelosis (Anaemia Spinalis, as in pernicious anaemia and other deficiency diseases). Initial pain occurs, sometimes of a radicular nature, in association with this malady, characterised by ataxia and paraplegia (Kinnier Wilson, p. 1353). Brouwer (p. 43) thinks that this pain is probably associated with neuritic processes, but states elsewhere (p. 40) that it is liable to be very severe (in the legs) and occurs *a long time before* further neurologic symptoms are presented.

Strümpel's spastic spinal paralysis and Little's disease. Although sufferers from these maladies were handicapped "by motor disability, overaction and muscular hypertonia", Kinnier Wilson (p. 783) does not mention complaints of pain. Does he advise Foerster's radicotomia posterior only to overcome the spasms? Brouwer (p. 62) refers to the occurrence of pain due to rigidity of the muscles. He does not do so, however, when describing the "contractures des arthéromateux of Démange". This does not appear strange to us, since these contractions in the lower extremities soon become so violent that movement (and, therefore, the inducement of pain?) is no longer possible.

Multiple sclerosis. We have already referred to the possible relation between multiple sclerosis and low back pain in Part I, p. 20. We found that 20% of the patients with an abnormal gait suffered from a weak or painful back without presenting the symptoms of hernia nuclei pulposi.

We would make the following comments with regard to an extensive investigation made into the correlation between low back pain and locomotor disturbances.

35 of the 150 patients examined had no locomotor disturbances. 24 altogether of the 150 were reported as complaining of low back pain (= 16%). *Only one of the patients with normal gait complained of low back pain.* Hence the percentage of *low back pain sufferers* among the 150 − 35 = 115 patients whose gait *was* pathological was 20%. On an average, the locomotor disturbances existed for 4½ years, with a spread of one week to 40 years. 21 of these 115 patients suffered from low back pain and in these patients the locomotor disturbances had lasted a far shorter time, *viz.*, 2¾ years,

with a spread of one week to 8 years. It is difficult to interpret the difference found. Assuming that low back pain in association with multiple sclerosis is caused by an abnormal static strain on the spine, quite conceivably bedridden patients no longer suffer from it, *i.e.*, patients who, as a rule, already have a long record of illness behind them. However, it is precisely these patients who, in their present parlous condition, are unlikely to refer any longer to "a bit of backache" they may have had many years before. In a word, the beginning of low back pain is plainly correlated with the onset of locomotor ataxia, pareses or spasticity. They often start simultaneously Sometimes the low back pain seems to be the first subjective symptom of multiple sclerosis and in three cases was, in fact, the only or chief reason for the visit to the doctor.

From fuller descriptions it would appear that the pain is usually chronically recurrent, or that there are remissions and exacerbations. The pain often radiates to one leg or both, but no records state it to be clearly radicular in character. Certain movements are liable to worsen the pain, more particularly stooping, but coughing, sneezing or straining seldom affects the severity of the pain in any marked degree. There is either no head-bending symptom, and the like, at all or else a mere trace of it.

Only a few lumbar spines were X-rayed. In one case clear arthrotic changes were to be seen and, in another, a prolapse of the disc. A trauma prior to the low back pain is mentioned in two cases Evidence of a traumatic aetiology of the low back pain, or of an abnormality of the spine as the cause of it, was produced in only a few cases. As against this, it is highly probable that the psychology of both examiner and patient is responsible for a disparity between the recorded incidence of low back pain and its actual occurrence. Patients in a pitiable state have something else to complain about than "a little backache", while the doctor, knowing from the documents that the patient admitted is suffering from multiple sclerosis, may not invariably add to his entries of diplopia, paralysis and incontinence the fact that the patient also has a certain amount of backache! As a matter of fact, when going through the available records, we had an opportunity of checking up on this here and there. Thus, in one case the low back pain was mentioned in the correspondence, but not in the clinical history, while in another case the low back pain was entered in the polyclinic records, but was not mentioned in the clinical history.

All in all, it seems highly probable to us that the figures given here for the incidence of low back pain in sufferers from multiple sclerosis are minima. Greater certainty on this correlation is likely to be obtained, not by the scrutiny of a yet larger number of clinical histories, but by questioning many patients with this correlation in mind

Abb and Schaltenbrand found among 1420 patients suffering from multiple sclerosis:

22.3 % back complaints
27.6 % rheumatic complaints without rheumatism
25.8 % disturbed sensation in the legs
27.5 % paraesthesia in the legs

They defined these subjective early symptoms as a pseudo-rheumatic (prelim-

inary) stage. The complaints were accounted for by radiculitides, foci in the sensory systems of the spinal cord or were ascribed to *spasm of the muscles*, which is felt as pain and occurs before signs of the pyramidal tract show up.

Kinnier Wilson (p. 157) does not mention complaints of pain as an initial symptom, but does in a more advanced stage, when spastic (para-)paresis and ataxia have developed. He then speaks of *pain in the arms and hands*, which is accompanied by paraesthesia, disturbed sensation, loss of finer motoricity and ataxia. He then proceeds to point out that, in addition to this cervical type, there is also a lumbo-sacral one which, in view of disturbed sensation and a lowering of reflexes, must be differentiated from a cauda-conus lesion. Patients complain of pain less commonly than of paraesthesia (p. 165); such pain may be indicated as girdle pains, sciatica, radicular pain and mislead the examiner into making a wrong diagnosis. The writer does not commit himself as to how such pains are brought about. Nor does he attempt to account for the occurrence of central sensations of pain, such as those associated with syringo-myelia, which are generally thought to be caused by lesions of the spino-thalamic tracts. Brouwer (p. 179) says that pain in the arms or legs and in the lumbar spine is a symptom of, mainly, the early stages of multiple sclerosis. Müller (quoted by Brouwer) believes that the so-called "rheumatism and influenza", which are often said to be causal factors, are simply the initial symptoms of this malady. Zukschwerdt (p. 185), speaking about something entirely different, refers to the secondary changes in the spinal column associated with multiple sclerosis and syringo-myelia. On page 98 he says that when a root is irritated, the possibility of multiple sclerosis, in particular, syringo-myelia and amyotrophic lateral sclerosis should be considered and ruled out.

Lewin (p. 49) mentions these same diseases as possible causes of low back pain. On page 608 he deals with multiple sclerosis separately in the chapter headed: "Neurological Lesions Affecting the Back".

Amyotrophic lateral sclerosis. Kinnier Wilson (p. 1016) states that this is quite often associated with paraesthesia and (muscle) cramp in the legs, though this is not pathognomonic for the disease. Brouwer (p. 150) says it is by no means rare for the patients to complain of pain in the back and extremities, sometimes, indeed, the pain is so severe that it may mislead one into making a wrong diagnosis.

In his monograph on this disease, Tans (p. 31), reviewing 87 cases, says that 15 patients experienced pain (as a prodromal, not as a pathognomonic symptom) in the extremity which shortly afterwards became *paretic*; 14 patients had pain, cramp and paraesthesia in other extremities. In a personal interview the author stated it as his opinion that a well-founded explanation

of these symptoms cannot yet be offered. It appeared to him that there were two possibilities, *viz.*,

1. Sometimes minor abnormalities are found in cells of the general visceral efferent system (usually primary irritant). These might account for vasomotor and other reactions taking place in the autonomic system.
2. Changes in the posterior cords are reported in association with this disease. Abnormalities are seen in the larger cells of the posterior horns of the spinal cord.

Scot (Spiegel 1957, p. 323) believes that sharp and burning pain referred to a lower extremity and unexplained by extra spinal findings, must be investigated for a possible intraspinal lesion. He reports six cases in which the explanation was found in tumors of the high thoracic and cervical parts of the spinal cord.

If we suppose, with Tans, that an extension of the pathological process through sensory systems in the spinal cord is responsible for the evocation of the sensations mentioned, we are still left with the strange and unexplained fact – likewise left on our hands in the case of multiple sclerosis – that by no means infrequently sensation, in the painful regions for example, is undisturbed.

Poliomyelitis anterior acuta. We shall not consider here the subjective symptoms which the patient experiences in the acute phase. The pain due to residual conditions (shortening of the leg, paralysis, atrophy, postural changes, such as scoliosis) was discussed in connection with the effect of such deformities generally. We may add that several authors have drawn particular attention to disorganised motoricity, or disorganised posture, brought on by residual malformations due to Heine Medin's disease (Prick, 1958, Epstein, p. 122 and many others). Brouwer (p. 66), too, draws attention to contractions and marked deformities of the back. We mention Brouwer again individually because, in his description of

Poliomyelitis anterior chronica, he involuntarily reduces the problem of the effect of disorganised motoricity to its correct proportions and throws considerable light on it. "At first", he says, "the patient gradually becomes aware of weakness in one or both legs. After a few *weeks* the legs become thinner and walking becomes increasingly difficult. By degrees the muscles decrease in volume and, sometimes in a few weeks, but usually in some months, all four extremities have become decidedly paretic". There is not a word about pain, contractions or deformities; only: "In severe cases, especially those beginning at an early age, paralytic contractions occur".

It is quite obvious that no secondary changes due to the disturbed motoricity develop in the *subacutely* paralysed sufferer from poliomyelitis chronica,

whereas the *chronic* effect of disturbed motoricity in the partially invalided Heine-Medin patient does produce them.

We may summarise our inferences as follows:

1. Pain, if any, produced by the majority of neurological maladies associated with motility disturbances is ascribed to the effects of the disease itself. The explanation of this is not always convincing, for instance when the pain is ascribed to a lesion of the pathways of pain, yet there is no interference with sensation.
2. It seems to us extremely difficult to judge, by reference to the particulars given in the literature, whether secondary pain occurs in association with motility diseases, if the authors in question have not been on the look-out for it. Tans (p.30) came to the same conclusion: "It should be added that the various investigators probably did not always pay attention to these *atypical* symptoms, which do not belong to the essential characteristics of the disease (such as pain)".
3. Neural disturbances are able to produce deformities (scoliosis, etc.) similar to those which, deriving from another cause, are known to be apt to produce disorders of the back and legs.
4. Complaints of pain are reported in association with a small number of the neural disorders which are accompanied by motility disturbances. The anatomical substrate of the disease provides no clear explanation for the pain experienced, nor do the various authors suggest any.
5. This pain occurs predominantly as an initial symptom in the prodromal stage.
6. The impression we have received is that disturbances in motility due to paralysis have greater effect than those resulting from ataxia. Perhaps a relevant consideration is that the latter group of patients are slow and deliberate in their movements and therefore move less, even if it be in an unusual way.

This review of the literature is incomplete and one-sided. We have not elaborated it because we do not attach much importance to it, for the reasons given above. An investigation of the matter made upon a large number of patients would also be disappointing, because it is necessary to take into account factors which are difficult to interpret statistically, like constitution.

Back complaints due to disturbed psycho-motoricity?

Useful indications are even more scanty in the literature. The question could be divided into two parts, *viz.*, (1) Is motoricity influenced psychically? and (2) If so, would the resulting disturbances be such as to produce symptoms?

To link up what has gone before with the material we shall now discuss, *i.e.*, neurologically with psychically disturbed motoricity, we quote some passages from Buytendijk's book, "Algemene theorie der menselijke houding en beweging" (General Theory on Human Posture and Movement). He himself considers it to be "a connecting link and contrast between the physiological and psychological approaches". The quotations are arranged in a way that makes them speak for themselves and calls for no explanatory comment.

"The structure of the nervous system and motoricity in an adult animal and in man are inextricably reciprocal" (p. 406).

"It is not the nervous system which can learn" (to make new movements) "but the *person*, partly by means of this organ, which is one of the available means and one of the limiting conditions for every co-ordination and reco-ordination" (p. 426).

"Investigations have shown beyond a shadow of doubt that the whole process of movement, even of simple movements, is controlled in the main '*centrally*', by which is meant that movement to the last detail is determined by the impulse proceeding from the *central nervous system*" (p. 442).

"A basic characteristic in the nervous system, which we shall call 'stability', may be postulated for one of the typical fundamental features of motoricity. Upon this will depend the manner in which all actions and gestures are performed. The factors on which the differences in innervation depend are tonic innervation component, extension, mode of generation, perseverance of the impulses and stability of condition in the nervous system; they themselves depend on the individual disposition and development and, therefore, on that complex of characters which is called the *constitution*. It is this, indeed, which, like the personal style of living, social standing and vocation, dominates the picture of an individual's motoricity" (p. 520).

"Any change in bodily proportions has an important influence on the child's habit of movement First of all, rapid growth is usually out of proportion to the development of the muscles, which means that movements call for greater exertion. Secondly, the plan of the body is changed. Even if, as a result, movements are less well co-ordinated in only a minor degree and there is consequently a certain amount of clumsiness and lack of control, strong subjective and affective repercussions will ensue in puberty." "The three characteristics of puberty, *viz.*, irritability, the disrupted *body-plan* and threshold position sense, give us an insight into the changes in motoricity which take place in this period" (p. 487)

"Corresponding to the 'break' in the course of life, we find the whole behaviour marked by instability, sudden changes, a number of typical con-

lictions. The failure of adaptation involves a lack of proper proportion, noderation, in everything, and this applies just as much to the distribution one at rest as to the force, direction and range of movements. The hyper- ;ric range of movement is no less obvious. The steps taken are exag- atedly long, several stairs are taken at a bound, both up and down, kick the furniture, hands upset everything, break everything. The lescent is *awkward and clumsy*" (p. 491).

[he same idea is expressed by Ruding, who points out that "the child has yet acquired an habitually automatic attitude". Lewin (p. 176) shares this nion: "Rapid growth is another cause of poor posture". Not inconceiv- y, in fact, the "growing pains" of young children may be due to imperfect ptation to a changed body-plan. On this analogy it would likewise be a usible postulate that there is a failure of adaptation in the adult when the dy-plan is deranged. This latter does not only occur on a neural basis, but o, and perhaps predominantly, on a psychic one.

Clumsiness is sometimes constitutionally implanted in the body-plan, as uld appear from something Buytendijk says (p. 528): "We can observe the msiness of people of athletic build in daily life. This clumsiness derives in rt from inappropriate accompanying movements, as has been demonstrated experiment". (References to the relevant literature on these experiments then given.)

It is outside the province of this book to enquire whether, in cases of so-called nctional aggravation of, or possibly psychic complaints, the concentration a conversion symptom upon a particular part of the body is due to a ychically caused disturbance in the body-plan. For instance, is the pre- enstrual back pain "real", *i.e.*, caused organically by congestion? Or has e unpleasantness associated with the imminent event caused the subject to so preoccupied with the womb and the back that these have become the int of least resistance for the reactions of *this unpleasantness* and other discom- rts, such as lassitude, to make themselves felt? It is a known fact that many omen do not complain of pre-menstrual distress, yet they also know the dvance signs of impending menstruation. Once women, who have otherwise ways been plagued by these pre-menstrual symptoms (*e.g.*, backache), are rprised by unexpected premature menstruation, these heralding symptoms ay henceforth not recur.

This example might go to show that pre-menstrual backache is, in part, etermined psychically. The view outlined above throws some light on the nanner in which the conversion is referred to the part of the body involved.

Let it be added that nothing abnormal was found in the epidural vascular

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We may *summarise* this review of the literature on disturbed motoricity as follows:

1. Motility disturbances due to persistent abnormalities of the extremities, or of the spine itself, are liable to produce back pain or radicular pain.
2. Little is to be found in the literature in support of the view that a neurologically or psychically disturbed motoricity is capable of precipitating secondary organic changes or disorders. It should be borne in mind, however, that the problem as such has not yet *ad hoc* been set in the literature.

The following questions remain open:

(1) Why is a departure from the "normal" pattern of movement pathogenic in one individual and not in another?

We postulate a constitutional factor; a predisposition would seem to depend on the existence or lack of sufficient spare room (spinal canal and foramina) and on the condition of the bone tissue (e.g. osteoporosis) and of the cartilage.

(2) Why is one motility disturbance pathogenic, whereas another, perhaps far more severe, is not, in one and the same individual?

We do not yet know which disturbances are especially injurious, though it is clear that it is those variations from the "normal" pattern of movement which bring the most pressure to bear on the spinal column, through an unfavourable balance between force and weight.

system in those cases when a laminectomy had to be performed just before or towards the end of menstruation.

Despite the growing scientific interest in psychosomatics, it is not surprising that very little should be found in the literature on the significance of a psychic disturbance in relation to the occurrence of so complicated and so ill-defined a syndrome as low back pain through the operation of disturbed motoricity. (*E.g.*, Cantor and Foxe: *Psychosomatic Aspects of Surgery*; Booy: *Psychosomatics*.) The subject of references to psychosomatic aspects of back disorders – Lewin devotes a chapter to them, p. 641 – is what is commonly called psychic back complaints.

Prick and Van de Loo and Prick, Calon and Van de Loo approach the aetiology of primary chronic rheumatism from the psychosomatic standpoint. (Cantor and Foxe, p. 144, quoting Cobb, Bauer and Whiling, come to comparable views.) Their investigations provide strong evidence in favour of a causal relationship with pre-existing neurosis. They do not mention any association between this neurosis and a causal disturbed (psycho-)motoricity connected with this rheumatism.

Hoelen (p. 193) states that prolonged psychic motility disturbances may lead to organic changes in the joints and to organic wasting.

Asthenia provides a clear illustration of a condition in which motoricity is disorganised. In all probability it is, in fact, responsible for back trouble, but since, at the same time, the tissues are of inferior quality, one cannot be certain of the true cause.

As a rule, the human mind is capable of assimilating observable facts and appraising them by the ordinary human standards of culture. Mankind's observations find expression in the vernacular. Expressions like "having no back-bone" and "letting oneself go" imply the general observation that a certain mood is associated with a certain posture. We cannot now trace who among our colleagues it was who said that "hernia nuclei pulposi reveals itself during a depression". There is a substance of truth in this. We believe that an existing back disorder can produce symptoms during a depressive phase of life. Not only will pre-existent complaints make themselves felt more acutely in a depressive mood, but we think it is probable that, by "letting oneself go" (mentally and) physically, (minor) disturbances really are exacerbated.

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PART III

FURTHER RESULTS OF POINTS OF ENQUIRY SET FORTH IN PART I

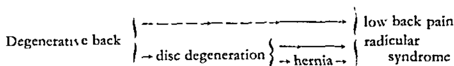
OUR OWN MATERIAL AND POSSIBLE INFERENCES

CHAPTER 6

SYMPTOMATOLOGY AND DIFFERENTIAL DIAGNOSIS

INTRODUCTION

Under some circumstances a degenerative back may be manifested by degenerative deterioration of the disc and herniation of the nucleus, though the syndrome likely to be presented consists of the components of three syndromes.



Thus the symptomatology of the weak back, combined with the chronic symptoms of the disc lesion, may be periodically accentuated by severe exacerbations of the hernia syndrome.

Quite apart from more exceptional superadded disorders, the clinical picture may be complicated by spondylarthrosis and radiculo-neuritis.

Primary or secondary spondylarthrosis } → low back pain
Primary or secondary radiculo-neuritis } → radicular syndrome.

Hence every case of low back pain and radicular pain implies the possible initiation of the complex of symptoms by a combination of several of the above disorders. It will be clear from this fact alone that, given this interrelated causality, it is difficult to define the exact symptomatology of one of these disorders

Clinical and therapeutic experience of hernia nuclei pulposi taught us a good deal about the symptomatology and the causes of low back pain. Although this study is mainly concerned with diseased backs to which hernia is (at most) incidental, we shall endeavour to illuminate the symptomatology of the degenerative back by comparing it with that of nuclear herniation.

Statistics were drawn up of the pre-operative symptoms of 500 hernia patients verified at operation. The results are expressed as percentages for the sake of uniformity and comparability with data from elsewhere. In 21% of these 500 cases, other disorders besides hernia (spondylarthrosis deformans,

knowledge of the symptomatology of nuclear retropulsion; he must also be well primed on the "surgically treatable back".

3. Lastly, "the relative value of negative findings" can be deduced from facts pertaining to unselected material. If, for instance, we find that in 75% of a series of hernias the patient experiences pain on bending obliquely backwards (Kemp), then this symptom is not pathognomonic for hernia, but neither is it for the disorders which were found in our material to occur simultaneously with hernia in 21% of the cases.

Unless otherwise stated, "hernia" in the following survey stands for an unselected case of hernia nuclei pulposi demonstrated at operation. By contrast, we use the expression "degenerative back" in reference to sufferers from back complaints and/or radicular pain showing signs of degeneration, though there is no reason to suspect, on the basis of the available data, a hernia nuclei pulposi. We do not possess exact information on the *incidence* of hernia, the degenerative back, complaints of low back pain and sciatica among the population. Lacking official figures, we cannot pursue an effective enquiry into the matter that would produce reliable information. However co-operative willing colleagues may be, no more than an indeterminate impression can be obtained, since the diagnosis is seldom established, several patients are treated together or consecutively by several or one doctor and many sufferers will not consult a physician at all. That the incidence is considerable is clear from a report ("Medisch Contact" 1952, No. 52) on absenteeism through illness in the Netherlands:

Through influenza	23.2%
„ diseases of the digestive organs	10.7%
„ *"lumbago"*	8.0%

In our statistics the incidence of hernia in *men and women* is about the same, contrary to the first reports:

	Women (%)	*Men* (%)	
Zaaijer	21	79	before 1943
Kostić	23.4	71.61	from 1948–1954
Mayo Clinic	28.4	71.61	before 1943
Krayenbühl	41	59	up to 1956
Ursula Clinic	47	53	up to 1954

It would seem as though, in the oldest series, the breadwinners received preference for treatment. The importance attached to the supposedly harder work done by men as part-cause of hernia seems to us exaggerated on these grounds.

gross manifestatios of status dysthaphicus, syphilis, T.B., overt anomalies, etc.) were found. Yet, after elimination, the symptoms of the remaining "pure" hernia patients (400) occurred roughly in the same percentages as in the initial material! In other words, the symptomatology of a series of selected hernia patients did not differ significantly from that in which several syndromes were intermingled, nor from that of the eliminated group. It follows from this that these syndromes overlap and this means that *a pathognomonic complex of symptoms does not exist either for one or the other back complaint.*

Although this did not make any practical difference, we had to decide whether to continue working on the series of pure hernias or on the unselected group. One's natural inclination would be to take the uncomplicated hernia cases, but there are three reasons for not doing so, *viz.*,

1. The data would then not be comparable with those found in the literature, in which no selection was made.
2. As investigations proceed, more and more particulars are collected which facilitate the task of selection. At first, for instance, one does not know the radiological signs; later one knows the composition of the cerebrospinal fluid; finally the abnormalities , which explain special aspects of the syndrome, are not seen, perhaps, until the operation itself.

The neurological examination and general impression (as a substitute for a thorough-going psychiatric examination) form the foundations upon which further investigations and the indication for them are built up. During the first contact with his patient, the examiner will probably weigh the chances of conservative treatment against the possibility and desirability of surgical intervention, even perhaps the admissibility of clinical observation. When other facts become known – usually under clinical conditions – a problem of a different order arises, *viz.*, defining the indication for operating. In the author's opinion, the complaint for which the patient is to undergo an operation then ceases to be the material point. Under certain circumstances he will wish to make the indication for an explorative laminectomy subject to the psychiatrist's approval. Even if there is no neurologically demonstrable symptom of an organic lesion and even if radiological examination and analysis of the cerebrospinal fluid show nothing abnormal, the patient has a right to the chance, at least, of being relieved of his disabilities by surgical treatment, provided his complaints are onerous, conservative treatment has been tried and the psychiatrist is satisfied that the pain is organic in character. The author believes that it is fundamentally wrong for surgical attack on low back complaints and radicular pain to be deferred until a hernia nuclei pulposi has been detected. For these reasons it is not enough for the examiner to be equipped with ready

Onset of herniation symptoms

62 % in the back. 38 % in one leg.
After back complaints, sudden onset of pain in one leg in 23 %, while in 8 % the back symptoms then ceased.

This provides a datum for differential diagnosis. Degenerative back symptoms are always first presented in the back. By degrees, symptoms may develop in the legs (pain, cramp, fatigue); radicular radiating pain seldom occurs suddenly then (neuritis associated with a chill) unless there is a hernia as well, in which event the back complaints do not cease.

The principal symptoms associated with low back pain can be grouped as follows:

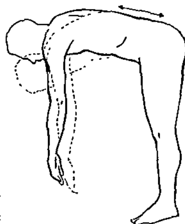
Mobility and postural disturbances.
Neurological symptoms.
Psychic symptoms.
Radiographically visible manifestations.

I. MOBILITY AND POSTURAL DISTURBANCES

Such disturbances of the back provide a clue to the condition of the intervertebral discs. We say that "the patient moves as though he has a disc lesion" if his back becomes rigid on flexion, if he has a permanent scoliosis, or acquires one on moving, and a corkscrew phenomenon occurs.

Associated with verified hernia

Rigidity	82 %
Scoliosis	52 %
Corkscrew phenomenon	77 %



This triad is not pathognomonic for hernia, the disc lesion *par excellence.* We have also encountered it in association with spondylarthrosis and the degenerative back without radicular pain. If a patient moves normally, it is very surprising to see a narrowed intervertebral disc on his radiograph notwithstanding; but one has to bear the possibility of an old "healed" disc lesion in mind.

Rigidity 82%. Let the patient bend forward with straight legs close together. There will be nothing unusual about the curvature of the back during the

On the other hand, we found that the incidence of back complaints *not* qualifying for operative treatment was greater among women (63%) than among men (37%).

After an extensive statistical enquiry into the incidence of all forms of "rheumatism" – including the complaints associated with hernia nuclei pulposi – de Blecourt likewise found that "rheumatism, in all its forms, occurs far more frequently in women than in men"; the occurrence of hernia (complaints) in men, on the other hand, was almost four times that in women.

In 1940 Kinnier Wilson observed back complaints generally, without any further differentiation, more often in men than in women.

Spurling (1958, p. 44), however, believes the incidence of hernia to be far higher in men than in women.

It has struck us in recent years that more patients suffered from radicular pain in the left leg as the result of a lateral hernia on the left (approx. 65%). By contrast, the *predilection for left-sided symptoms* in our first series of patients proved to be not more than 55%!

Similar findings occur in Waris, p. 34:

Malmrose	62%	on the left more than on the right
Yaskin *et al.*	58%	
Petis-Dutaillis & De Sèze . . .	59%	
Waris	51%	

In 52% *remissions* of the complaints had preceded the hernia operation. 100% of the degenerative back cases, in which we had verified at operation the absence of any abnormalities (*e.g.* hernia) other than the signs of degeneration observed, had passed through remissions, because surgical intervention was resorted to only if recurrencies continued, despite conservative treatment.

The symptoms of severe spondylarthrosis may abate temporarily, but it is never cured.

Duration of complaints	< 1	1	2	3	4	5	6	7	8	12	14	16	18	19 (years)
before hernia operation	33	11	10	8	4	2	2	3	3	2	3	3	2	1 (%)

The series of recent years includes some patients with a prolonged case history; but there is a noticeable tendency to ask for surgical intervention six weeks after abortive conservative treatment or immediately upon the first recurrence of the symptoms.

All the patients upon whom we operated for a degenerative back, without demonstrable herniation, had been suffering for more than a year, this being the minimum period set to assess the results of conservative treatment.

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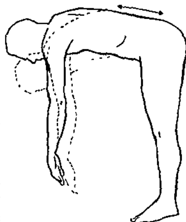
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complaints, stretched the 10 cm line to more than 14 cm, as is shown in the diagram (p. 256).

Spinal rigidity may be either accompanied with pain or not. Some patients cannot bend more than a few degrees, which would seem to point to a mechanical cause of the fixation in those cases. More commonly, however, the patient is capable of further flexion, but is inhibited by the pain it causes; we then consider the fixation to be a protective mechanism. It is fair to suppose that, during forward flexion, a diseased intervertebral disc is subjected to greater pressure, or the foramina are locally narrowed. We seem to have noticed, though we cannot express our findings in significant statistics, that mechanical stiffness is more often associated with a narrowed intervertebral disc, while the patient whose disc has not narrowed, or only very little, is prevented from bending by the pain.

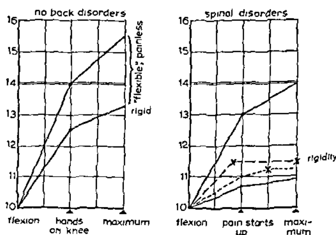
Any appraisal of lumbo-sacral stiffness should take account of the possible *rigidity* of the patient. (Age, inactivity, constitution.) There are some people who, though they have never suffered from back pain, hold their spine so stiffly that one suspects a locked back (see the diagram). But, on closer inspection, it is found that this stiffness is not confined to the lumbo-sacral level. The spine is also stiff to some extent for a varying length of time in the *post-operative* period.

The back can quite suddenly become completely locked and just as *suddenly* be freed. Usually these patients are able to bend further forward, but cannot straighten up again and experience pain when trying to do so; however, this kind of rigidity strikes the observer as being purely mechanical. We are dealing with these mobility disturbances elsewhere.

A lumbo-sacral fixation can also be *simulated*, or be of an otherwise psychic nature. Guidance for differentiation can be had from the following tests. The patient who, ostensibly, cannot bend far forward while standing is found to be able to raise the trunk quite easily from the supine position; if one helps him by holding out one's hands to him, the spine will flex in the same way as it would with the normal back in the standing position.

Burns (quoted by Lewin, p. 642) tested these patients in the following manner. They were asked to kneel on a low stool and then to bend until the outstretched arms touched the floor. There are no neurological nor mechanical reasons why the organically fixed back should not be able to bend far enough forward for the hands to reach the floor; this has been proved by the ability of patients with organic disorders to carry out this test. In cases of hysteria or malingering, the patient pretends to be unable to perform this movement. After having bent forward a few degrees, he refuses to let himself go any farther and this is a

initial movements. On further flexion, the lumbar-sacral back is held rigidly, while the upper part continues to bend to some extent. This itself shows that the rigid back cannot be roughly measured by the depth of flexion of which the patient is capable. A proper impression of the degree of rigidity cannot be obtained by estimating it by the ability or otherwise of the patient to touch the floor with his finger tips (Krayenbühl); despite marked lumbo-sacral stiffness, the upper part (if, for instance, the patient has a rounded back) may be able to bend so far that the fingers nevertheless nearly touch the ground. It is necessary, moreover, to make quite sure that the patient keeps his legs close together; otherwise the whole stiff back will bend in the hip joints. It might be useful to have an objective gauge for spinal rigidity in the matter of insurance policies and so forth. Smeele, formerly assistant at the Ursula Clinic, worked out one method. He placed a lath beside the rigid lower lumbar vertebrae and judged the degree of stiffness by the angle formed between the flexed back and the lath. This method had several drawbacks, one of which was the subjectivity of the assessment. We therefore adopted a different technique and tried it out in practice. We have since learned from Brocher's published paper (1957, p. 6) that Schober had already applied this method. A measured line 10 cm in length is drawn on the lumbo-sacral part of the spine, the top point being on the line joining the two pelvic crests. We used

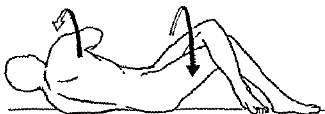


to mark this line with iodine, but later found it more practical to mark the two extreme points in ink. When the patient bends, he stretches the skin of the lumbo-sacral part of the back, the greater the flexion, the more the skin stretches. It transpired that patients moving "normally", without back




Detached vertebral arch

We found pain associated with *backward movements* to be almost pathognomonic for a detached vertebral arch; however, the same symptom is produced by pseudo-luxation.



Exacerbation of local paravertebral pain through *rotation* of the body points to a lesion of the vertebral arch.

The patient's *sideways twist on backward flexion*, as described by Kemp, is not pathognomonic for any of the back complaints referred to above. We found this to be positive in 75% of cases. The pain is brought on by forcing the back against the fixation and against the scoliosis. If this manoeuvre is applied to the patient who has no scoliosis, the test is less likely to be positive. It is our experience that the test may sometimes be positive homolaterally, sometimes contralaterally, without preference for the diseased side, and is liable to alternate in successive stages of the disease. The same happens with scoliosis. This would seem to substantiate the supposition that in this manoeuvre pain is initiated by movements parallel to or in the contrary direction to a scoliosis. The fact that the supposed scoliosis is not always seen does not refute this view, since a scoliosis may arise towards the end of a movement, be absorbed by protective mechanisms and vanish.

Scoliosis 52%. Scoliosis is a postural abnormality which may occur when the patient is at rest, standing and associated with movements. The patient may not be conscious of it. In other cases he may point out that he walks crookedly or that his hip is becoming "fat". Movements may either intensify or reduce the scoliosis, it may alternate from the right to the left and back to the right,

sign that the rigidity of the spine does not derive from organic disturbances. Then again, locking and stiffness of the spine should not be confused with the condition which the patient is trying to describe when he states that he feels so painfully stiff after resting or on rising in the morning. This is often associated with spondylarthrosis.

This *painful morning stiffness* provides us with an indication for differential diagnosis, in that it soon diminishes or disappears altogether after the patient has begun to move about. In such a case, therefore, we speak of a "*loosening-up symptom*" pointing to spondylarthrosis. By contrast, the patient suffering from a disc lesion feels rested in the morning and the associated pain in the back after movement and exertion becomes worse as the day goes on.

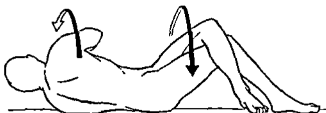
Many suggestions have been advanced to account for the mechanism responsible for nocturnal pain and pain brought on by movement after rest, but none is altogether satisfactory (Lewin, p. 46). Elsbach supposes that it is a similar mechanism to that operating in intermittent claudication: when the patient begins to move, the arthrosis demands more energy – *i.e.*, more oxygen – than the minimum available oxygen in the muscles of the supine patient. This, we think, is in itself disproved by the fact that even passive movements are then painful. It is also hard to understand that the muscles should need as long to make good the shortage of oxygen as the time usually elapsing (5 minutes to hours) before the initial pain dies down while movements continue. In view of the ankylosing proliferative process of arthrosis, a simple mechanical explanation appeals to us most, namely this: during the period of rest, the joint surfaces lack the grinding action of movement. As a rule, prolonged rest, *e.g.*, several weeks, initially alleviates spondylarthrotic complaints, but, on the principle that "rest rusts", the condition of the patient when again mobilised to some extent is worse than it was before.

Contrasting with the painful check on bending, there is the pain associated with *extension from flexion* of which some patients complain more especially. This likewise points to a possible spondylarthrotic condition; when describing this low back pain, the patient usually rests his hand against the lumbar region The symptom comes on after the patient has had a long rest, especially if he has to get up from a deep-seated arm-chair. It is not pathognomonic for any abnormality of the back in particular. It occurs in 58% of hernia cases Nor does the *hyper-extension* test produce clear indications In 62% of hernia cases it produces pain or exacerbation of pain. The hyper-extension test should be distinguished from bending backwards from forward flexion. The person tested may be able to bend forwards almost horizontally without any trouble, but any backward bending may produce pain in the back.



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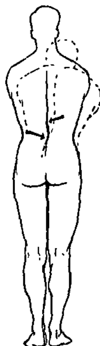


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without causing the patient pain. This obliquity of the spinal column, which can be seen on radiographs, may either be mechanical in origin or derive from contractions of the muscles. The mechanism can scarcely be ascribed to an unsymmetrical narrowing of an intervertebral disc only, for, with the same radiographically detected narrowing, one may see a scoliosis to the right or to the left, alternating with brief intervals. A more satisfactory view would be that the scoliosis is a protective mechanism against pain: if pain is caused by local pressure, a compensatory scoliosis to one side may mitigate the pain, while pain as the result of space limitation – *e.g.*, compression of a root in the foramen – can be prevented by scoliosis in the other direction. But it still remains difficult to accept painless scoliosis, of which the patient himself is unaware, as a protective mechanism against pain.



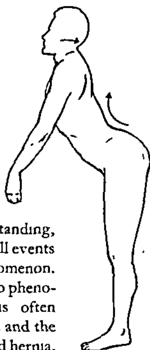
Corkscrew phenomenon 77%. This is the term given to a slightly jerky movement of the back which occurs on extension. The movements are then spiral like the pitch of a screw, as it were, and the patient is said to "corkscrew". This, too, can be regarded as a protective mechanism which adapts itself during movement to the changing conditions of the intervertebral disc caused by the movement. This movement is a resultant of partial locking and alternating scoliosis. It is liable to be almost unnoticeable and becomes more apparent if the patient is made to move quickly and, another time, when the patient is asked to straighten very slowly from a stooping posture. It is difficult to account for this phenomenon on merely mechanical grounds, but probably mechanical factors are combined with pain-resisting protective mechanisms by muscular contractions. We have tried, but failed, to get patients clearly subject to the corkscrew phenomenon to straighten out.

The corkscrew phenomenon is not to be confused with so-called *alternating scoliosis*, since this, to one side or the other, can also be seen when the patient is at rest; the movements, moreover, are far more pronounced.

There is also an *hysterical corkscrew phenomenon*, which is seen when the patient coils, as it were, while rising to the erect posture. It does not resemble the alternating type of scoliosis, which jumps from one side to the other; still less the finer nuances of the almost unnoticeable corkscrew phenomenon. It is as though the hysterical patient deliberately makes this exaggerated movement to impress his disability upon the onlooker. When he is asked to

raise the trunk from the supine position, he "forgets" to make this exaggerated movement.

Finally, the corkscrew phenomenon should not be confused with the symptom often seen in association with spondylolisthesis. When rising erect, the patient "*worms*" his back upward, as it were; in cases of normal back locking, the rigidity of the lower part of the spine is gradually overcome during this effort. The patient suffering from vertebral displacement first raises the upper part of the spine, the lumbo-sacral part continuing for a time to be locked. For a moment, the back then looks as though it has sagged; the head leans backwards and the buttocks project. The patient works off the listhesis to a lordosis, extends the hollow back fixed in hyperlordosis and, in almost the erect posture, neutralises this lordosis. When the patient is standing, the back is finally extended, sometimes jerkily, or at all events rather abruptly, and thus simulates the corkscrew phenomenon. In point of fact, it is not just by chance that these two phenomena resemble each other; for, spondylolisthesis is often accompanied by narrowing of the intervertebral disc and the same applies to an advanced disc lesion or well-defined hernia. We regard both phenomena as protective mechanisms to balance out excessive pressure on the spine or disc during extension of the back.



The corkscrew phenomenon need not occur if the back is very rigid and considerable scoliosis persists in the erect posture. (Fixation + +, scoliosis + +). The fixed scoliosis then prevents finely graded movements from being made. We would admit the corkscrew phenomenon as being pathognomonic for a disc lesion, provided it be borne in mind that the symptom is liable to be masked or withheld by the above serious disturbances of mobility.

The locked back. (Incidence?). The patient actually feels as though his back were literally locked. On bending down, he finds that he cannot straighten out again. This may happen after having performed some particular movement awkwardly, exerting force or lifting in a stooping posture. Yet there are cases in which the patient has done nothing worse than stoop in a manner he has done countless times before. He is often able to stoop further still, but cannot straighten up again. An "attack" of this kind is sudden, sometimes without – but predominantly after – a history of recurrent back complaints. The onset

of the "attack" is invariably stated to be very painful; straightening up is mostly painful, but we have come across patients who were quite incapable of extending their back, as though prevented from doing so by some mechanical obstacle, yet experienced no pain in their efforts to overcome it.

Radicular pain may occur. As a rule the Lasègue test is not positive until the stretched leg has been raised to the full limit.

Unlocking is generally a gradual process; quite often a patient cancels arrangements to be admitted to hospital because his back has reverted to normal. Sometimes, too, a given movement will suddenly release the back. When another person suffering from a locked back hopefully imitates that movement, it is usually in vain.

The chiropractor applies manipulations which may unlock the back and bring relief from pain, but the results of this treatment are unpredictable and many patients suffering from a locked back derive no benefit from it.

On very rare occasions we have operated for hernia while a back was locked. After the laminectomy the back was unlocked. Nothing unusual was found during the operation that would account for the locked back.

Explanation of the locked back

a. Unilateral hyper muscle tone. E.g., after catching a chill, or resulting from unilateral painful contractions initiated by diseases of other organs (Biedermann, Mackenzie, Zukschwerdt). These conditions, however, are not related to the locked back; there is no unilateral hyper muscle tone; furthermore, the internal disorders will be associated with other symptoms. Back-locking, on the contrary, is preceded by periodically recurrent back complaints.

b. Locking of the diseased or healthy joint by *joint-mice, subluxated joint surfaces* or interarticular discs, or *involvement* of sequestra of the nucleus pulposus (Junghanns, Rasch, Zukschwerdt). The acute onset of the symptoms, as well as their acute disappearance in response to manipulative treatment, give some colour to these suggestions. Moreover, joint-mice (torn-off joint facets) and haematoma in the discs have been found (Zukschwerdt); but pseudoluxation of a lumbar intervertebral joint has not been seen. Patent joint fissures have been recorded by radiographers, but they also see wide intervertebral joint fissures associated with backs that are neither painful nor locked. In this matter we share Junghanns' opinion that more than one factor is involved in back-locking. We should, however, not overlook the fact that "all" these locked backs have records of antecedent periodic back trouble (!) With this in view, we also subscribe to Zukschwerdt's postulate as regards the mechanical locking of the intervertebral joints. He believes that an antecedent

chondrosis makes the vertebral joint vulnerable to subluxation. We have two alternatives in mind, *viz.*,

The chondrosis brings about changes in *the same* segment, *e.g.*, in the joints through narrowing of the disc and the often associated slight posterior displacement of the vertebra above this disc.

The mobility of the segment involved is disorganised by the chondrosis. Upon flexion of the spine, greater than usual amplitude of movement is required of *other* mobile segments to compensate for the failing chondrotic segment. (See functional listhesis, p. 438).

c. As Lewin states, each segment contributes a small share to the total mobility of the vertebral column. It is in this light that we have to consider the neural therapist's postulate (Gutmann and others) that one small local lesion is a *"vertebral field of disturbance"*; at a distance, it checks or blocks the mobility of several co-operating segments. It can be compared to spasm occurring in the collateral circulation of a thrombosed blood vessel (following Leriche).

Our experience in this matter is limited. All we know – through personal observation while with Huneke, Dusseldorf, 1955 – is that, without restorative manipulation, a locked back can be relieved of pain and made flexible again by injecting a small quantity of Impletol in a so-called distal focus or in the spinal column. Huneke himself refuses to treat a patient who, either in his clinical history or in recurrent attacks, presents the syndrome of a hernia, as "hernia cannot be cured by neural therapy" (statement made personally to the author).

d. Concealed disc. In the Anglo-Saxon literature it is held that a nuclear sequestrum may possibly bulge through the annulus fibrosus and subsequently slip back again. (Dandy, Falconer, Lewin and many others.) Armstrong describes the process in more precise terms, assuming that the nuclear sequestrum becomes impacted between the rims of the vertebral body, through which the segment involved becomes locked.

Several objections can be raised to this view. Mechanical locking of one segment of the spinal column reduces its mobility, but cannot be accountable for the inability to straighten out the whole spine. As we know, the initiation of pain in one segment does not generally lead to locking in association with the other back lesions. Besides, some forms of locking are not painful (!)

Nor can we see how a nuclear sequestrum can become impacted exactly at the point of rupture, seeing how great are the forces brought to bear upon it during movements. It is easier to imagine the nucleus as being shot out, either forwards or backwards, like a cherry-stone. Retropulsion does not seem to occur, or to do so rarely, as the patient is usually symptomless after the back has been unlocked, or at any rate shows no signs of the root

compression which would in that case very likely occur. Rapid displacement of the nucleus in the ventral direction – in other words, the return of the extruded nucleus into the disc – seems to us a mechanical impossibility as an acute incident. It does, we believe, occur in a chronic stage; it is thus that an old hernia spontaneously "heals"; the nucleus and the remains of the disc have had time to change, *i.e.*, to degenerate further or to have become necrotic. Back-locking through the re-entry of a *fresh* nucleus, or its being "sucked inwards by a favourable movement", seems to us to be impossible, because it conflicts with the accepted idea of the mechanism of an intervertebral disc. This disc stands under tension, a tension which *extrudes* its contents through a breach. For this reason any attempt at operation to push back a freshly extruded nucleus fails. The tension in the disc is maintained by the tissue turgor of the intact intervertebral disc; as soon as the disc ceases to be under tension, the exchange of nutrient body juices is lost. It is not only the nucleus which escapes through the breach, but the life from the interior of the disc with it. The apparatus collapses like a punctured motor tyre (Lewin). The nucleus and adjacent structures receive no further nutrition and become necrotic or degenerate to a mucoid substance. We presume that a concealed disc can occur in this chronic condition. At operation one finds a collapsed, soft disc, the contents of which are mucoid. The breach is closed by that time. Quite often the root has grown over this "healed" hernia. Extruded nuclear tissue takes the form of a necrotic mass, both inside and outside the closed breach, in the latter case usually in a favourable spot somewhere within the spinal canal where it no longer affects the cauda.

Examples are known of a root adhering to a secondarily closed breach, while the extruded nuclear sequestrum had migrated under the contralateral root, there causing "hernia" symptoms. We believe that necrotic matter of this kind can shift up and down at the site of the breach before this closes. It seems all the more inconceivable to us that this soft substance would be capable of offering resistance to the immense forces brought into play by movements, in this case extension of the back

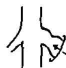
e. Locking is ascribed to *oedema* of root or adjacent structures (Thurel and others) Oedema can come on rapidly. It might be reasonable to suppose that, through lack of room at a given moment, the systems are squeezed acutely. But it is more difficult to imagine the back's suddenly becoming freely mobile again on account of abrupt withdrawal of the oedema.

Further objections are Usually a back remains locked after a sudden awkward movement; could this abruptly induce oedema? We have not been able to discover such an oedema of roots, ganglia or adjacent structures;

exact data are not to be found in the literature, though several authors state frankly that the occurrence of oedema is conceivable, but has not been proved. Anyhow, it is still an open question whether this supposed oedema could occur so *suddenly* as the result of some unfortunate movement.

The back becomes locked upon *flexion*, because it is after this that the patient is unable to straighten up. In the Lasègue test, the roots are pulled towards the foramina during flexion. In the process of extension, the roots shift in the opposite direction. These, together with the ganglia, are thickest at the entrance to the intervertebral canal. If we ascribed back-locking to the pinning down of an oedematous root, it would signify that the thickest part of the supposedly oedematous, swollen root could not shift from the narrow space just in front of, or in, the foramen to the wider space in the vertebral canal. This is an absurdity! No less fallacious is the assumption that an oedematous, swollen ganglion is pulled by an awkward movement in the stooping position into the canal and is clamped there. For, in the Lasègue test, the maximum amplitude of movement of the shifting root is 2 to 8 mm; add to this that the displacement of the roots associated with stooping is scarcely comparable with that occurring in the Lasègue test, since, unless asked to do so, no one bends as far as possible with legs kept straight. Moreover, the person with a trapped ganglion would suffer continuously from very severe pain, such as that induced by the Lasègue test. Nevertheless, in some rare cases of locked back with accompanying continuous radicular pain, a swollen ganglion might possibly get jammed in a constitutionally narrow lateral recess of the spinal canal when the patient stoops.





We are inclined to think that the back can become locked when the bobbin-shaped, tapering, thicker part of the root and ganglion is "trapped" behind a prominence in the spinal canal while the patient is stooping. Such a prominence can be formed by the stooping action itself; we have in mind a protruding disc It may be that, during the movement, the thicker part of the root and ganglion is pulled between the pedicle and an already existing protuberance, *e.g.*, a hernia, the rim of a bone or an injured joint. This would account for:

Recurrent back [illegible]

Sudden release of the back.
Pain on trying to straighten up
No pain as long as the back is stooping.
The ability to bend down further painlessly.

That this (neurogenic) explanation is probably correct can be demonstrated by the Lasègue test applied to someone with a locked back. It is difficult to perform, because the supine patient, lying on his locked back, keeps his legs bent. Raising both legs relieves the back, provided the patient relaxes the back muscles. In the event of radicular pain, we noticed that there may be an intermediate position in which the raising of the stretched homolateral leg alleviates the radicular pain. We were unable to alleviate this pain by raising the contralateral stretched leg and found that it increased the radiating pain. If it is a fact that the contralateral (crossed) Lasègue displaces the captured root from the foramen to the middle of the spinal canal, then the latter test proves our case.



In point of fact, we are inclined to agree with Junghanns that the back can probably be locked through the agency of several mechanisms. It would seem that the painless fixation, which makes the impression of being a mechanical one, is caused predominantly by some disorganisation of the joint. If extension is checked by pain, it is more likely that nerve tissue is being pinched.

The multiplicity of suggested explanations is partly due to the fact that different authors approach the problem of the locked back from different angles. Lewin, for instance, associates it with a symptomatology which we should regard as a typical "hernia" attack. Then it is our impression that chiropractors use the terms "joint rigidity" and "vertebral joint locking" in a more general sense; they also include sudden attacks of pain which check the mobility of the spine or are associated with a scoliosis. As generally understood, a locked back denotes the sudden inability to straighten up from a stooping position.

Other symptoms which can be regarded as mobility and postural disturbances are fairly well known and no useful purpose would be served by enumerating them all, but we may mention the tendency to sit in a crooked posture, constantly shifting from one buttock to the other; sitting by preference in a straight-backed chair; bending the knee when stooping or sitting upright; or some particular habit in bed, such as preferably sleeping with drawn-up legs, or flat on the back, and so forth.

These tendencies have no pathognomonic significance. If the complaints have led to these habits in a short space of time, the odds are, to our mind, that an organic disorder is at the root of them, since a psychically disturbed person does not "know" these signs.

II. NEUROLOGICAL ASPECTS

The neurological symptoms are often the basic indications for clinical observa-

tion or some form of treatment. Sometimes they form the basis for the granting of allowances and they are also interpreted in statements on occupational suitability or otherwise.

It is all the more disappointing to have to admit that the neurological picture of the patient with back complaints and radicular pain provides no symptoms of real pathognomonic significance and that a symptom seldom offers a datum for differential diagnosis between the various disorders. Lastly, the interpretation of the facts discovered at neurological examination is unreliable, also for locating a lesion.

Lasègue test +

	Hernia n.p. L.IV – L.V and L.V – S.I			Hernia n.p. L.III – L.IV
Ursula Clinic:	94%	46% also crossed	5% only crossed	60%
(Krayenbuhl:	96%	60%)		

The Lasègue test is performed by raising the supine patient's stretched leg. If the test is positive, pain occurs as a rule when the leg has been raised more than 30° from the table on which the patient is lying. The height at which pain is felt varies from individual to individual; so the examiner often records the angle which the raised leg has reached at that moment. In our opinion, so many other factors are involved in the Lasègue test that it is futile to let the symptom depend on the size of this angle. In the event of a constitutional rigidity, the picture is obscured by an admixture of other complaints. One has to question the patient as to whether he feels pain in the hollow of the knee, as some patients, in whom the symptom may be assumed to be negative, are prone to the sensation of pain through over-stretching of the muscles. We do not attach much value to Bragard's sign, as a complement to the Lasègue, if the pain does not radiate. Under the Bragard test the pain may remain localised in the calf, and if so, this pain, too, must be distinguished from over-stretching of the calf muscles.

We found the Lasègue test to be positive in conjunction with every condition pointing to a lesion of the radix or the peripheral nerve, such as radiculitis and neuritis. Not only was the symptom positive in our experience in association with hernia verified at sight, but also when no hernia was found in a so-called "negative exploration", with anomalies of the cauda and the dural sac, while spondylarthrosis also appeared to be able to produce a positive

Lasègue if accompanied by narrowing of the intervertebral foramina.

Reischauer, noting that the Lasègue is less commonly positive in association with herniation of the L.III – L.IV disc, suggests that, through straightening the leg, the sciatic nerve, the main component fibres of which derive from the S.1 and L.5 roots, is stretched. It is the L.3 and L.4 roots, on the other hand, that supply most of the material for the femoral nerve and this nerve, which runs from the plexus to the anterior part of the leg, is relaxed rather than stretched in the Lasègue. Plausible as this suggestion may be, we have to remember that the Lasègue associated with hernia of the L.III – L.IV disc was positive in 60% of the cases! It may be inferred that enough fibres run from the L.3 or L.4 root *via* the sciatic nerve to set up pain in the relevant case through over-stretching. Nevertheless, it is important to bear in mind for the diagnosis of "hernia nuclei pulposi of the L.III – L.IV disc" that the Lasègue often remains negative in this localisation.

The Lasègue is a reliable symptom if the pain induced radiates into the leg, and preferably in the same way in which the patient previously experienced it spontaneously. We then call the Lasègue *neurologically positive*. Sometimes, too, on straight leg raising, the pain is stated to occur only in the back; it is not clear to us yet whether in that event the Lasègue is to be interpreted as being positive. That is why we differentiate between "neurologically positive", when the pain is felt in the leg, and "positive" when pain is felt anywhere. On raising his leg, the patient, expecting pain, keeps the pelvis rigid; the pelvis is therefore involved in the manoeuvre and we surmise that the pain thus initiated is due to a change in the position of a (diseased) intervertebral disc caused by the moving pelvis. Thus the pain in the back accompanying the Lasègue test probably has its origin in movements of the spine and, as such, is not specific for the mechanism of pain through nerve stretching which is ascribed to the Lasègue test. The correctness of this view can be confirmed by fixing the pelvis to the table, when pain is no longer felt in the back during the test, alternatively, the test proceeds painlessly, or else results in a neurologically positive Lasègue with pain only in the leg.

Hence the first thing to watch is the pelvis *i.e.*, whether it is held rigidly or not. An almost identical "trick" is the socalled *"acquired Lasègue"*. There are quite a few patients who, having been examined several times before, know in advance what straight leg raising involves. They stiffen themselves, maybe without indicating pain The examiner ecounters great rigidity, which cannot be broken down and he might soon be led to interpret such rigidity as a positive Lasègue symptom. There is also the *Lasègue reflex*, *i.e*, when the patient passively allows the straight leg to be raised but, upon the least hint

of pain, becomes taut. Depending on the patient's nervous excitability and over-sensitiveness to pain, this sudden tightening of the muscles is accompanied by exaggerated indications of severe pain. Obviously, these various manifestations cannot all be taken at their face value as a sign of root or nerve stretching. It is, furthermore, necessary to take account of *constitutional stiffness*, which varies from individual to individual. One certainly should not expect to raise the straight leg of elderly people to 90° painlessly. One sometimes sees, to one's amazement, robust examiners performing the Lasègue test in a manner which could scarcely fail to cause pain to anyone. Lissome individuals possessing a sound muscular system, who have kept themselves in good general condition by physical exercise, will allow the leg to be raised beyond 90° without signs of pain. As against such people, others with a natural stiffness of the flexors in the hollow of the knee will not be able to bear such treatment. To our mind, a better procedure to obtain a reliable indication of this symptom is first to raise the leg several times with the knee bent, then to try out how far the leg can be stretched from the knee without causing pain. Once it is outstretched as far as possible without causing pain, the leg should be raised until pain is felt. After all, the point is the presumed stretching of the sciatic nerve, not the absolute degree of pain.

The painless extensibility of the sciatic nerve of the person who moves with stiff leg muscles will normally be relatively slight, and the object is to measure that extensibility.

By the "*reversed Lasègue*" we mean stretching the straight leg in the hip. The patient lies supine on the edge of the examiner's table and the straight leg is moved downwards from the edge of the table. Very occasionally this test was positive, notably in those patients who complained spontaneously of a radiating pain in the groins, which made us suspect a hernia at L.III – L.IV. It is difficult to account for the fact that pain occurred when we *suddenly let go* of the stretched leg during the Lasègue test. When we had raised the leg to, say, 60° and then let it fall, or moved it abruptly towards the table, the patient sometimes suffered for a short time exactly the same pain that he felt spontaneously. This cannot be due to real stretching either of the sciatic or the femoral nerve. The accompanying pain in the back is presumably caused by sudden muscular tension; the brief stabs of radicular pain may possibly be brought about by the mechanism to be described in the ensuing pages.

Finally, we have to distinguish between a classical positive Lasègue and the feigned, exaggerated pretence of pain on the part of the *malingerer* and the *hysteric*. Experience is needed to assess the pain reactions at their true value; the examiner should not be biased in his judgment of a patient suspected of

having this kind of mental make-up. Moreover, as has already been hinted, sometimes the patient has been examined time and again and knows that straight leg raising is expected to cause pain in suspected cases of hernia or some other lesion of the back. This approach on the part of the patient can be circumvented by making him perform movements which, although he is not aware of it, are an imitation of the Lasègue test. He is told to bend forward, if he does so with some rigidity, he is made to take a few steps in that position with *straight* legs. In doing so he is actively carrying out almost the identical movements which are made passively by raising the stretched leg. In this way the Lasègue can be gradually imitated by making the patient take steps of varying length. We have found on several occasions that the same patient who stated that slight raising of the leg in the supine position was painful, was able with impunity to take large steps while stooping and keeping his legs straight.

What can we suppose the mechanism to be which causes pain during the Lasègue test? Let us take the case of a patient who, on straight leg raising, typically feels the same radicular pain which he has sometimes experienced spontaneously in the same place. Movements of the spinal column and associated protrusion of an injured disc were prevented by immobilising the pelvis during the test. We know that the root shifts from 2 to 8 mm when the straight leg is moved (Schmorl, Krayenbuhl), as Sjöquist demonstrated in an operation film (Madrid, 1953); we have actually seen it happen several times by performing the Lasègue on the operating table. The root shifts in this way both when it is compressed, say by a hernia, and when perfectly free (Thurel, Zukschwerdt). We have seen the root move visibly in cases of a positive as well as of a negative Lasègue, with a hernia and without one. The inference from these summary facts is that something out of the ordinary must occasion radiating pain in association with the root movement. It may be an obstruction in the vicinity of the root, such as a hernia, lipping or a narrowed foramen; or there may be changes in the root itself (compression or adhesion, primary or secondary thickening oedema, radiculitis).

In the literature, pain associated with the Lasègue test is explained as follows.

(a) Pain is produced by stretching of the roots, etc. (Spurling, 1958, p. 54, De Sèze *et al.* 1957, p. 1013) In their postulate respecting the ipso- and contralateral positive Lasègue test, the latter authors do not take account of the distension of the roots just in front of the ganglion.

(b) Pain is produced by the inhibition of movement by root adhesion or root compression.

(c) Pain is produced because the movement involves a root irritated by inflammation or oedema.

If we apply these explanations to a patient who had monoradicular pain pre-operatively and in whom a hernia nuclei pulposi was verified at operation under the root, this, when irritated, causing the pre-operative monoradicular pain; if, moreover, we know that the Lasègue test produced identical monoradicular pain, it is not always possible to reconcile the suggestions put forward in the literature with these facts. This because it has been established beyond doubt that the Lasègue test was also positive in cases where nothing abnormal at all was found in the course of the extradural root (no hernia, no oedema, no radiculitis and no anomaly). In such a case the origin of the pain irritant as the result of movement of the root has to be sought peripherally to the extradural root. This might be the ganglion, the plexus or the peripheral nerve. The radiation of pain having been typically monoradicular and knowing that disorders of the plexus and the peripheral nerve do not produce monoradicular disturbances, we have only the ganglion and most proximate structures left that could transmit pain with movement of the root in the Lasègue test.

It is apt to recall here that the root in its extradural course is as smooth as a tube from the dural sac to a distance of several millimetres, but then thickens, like a bobbin, until it enters the foramen, where the ganglion is situated. In taking account of this fact, it should be recognised that the thickest part of the root is displaced by the Lasègue test towards the foramen, hence in the majority of cases *away* from an existing hernia, if any. We have evidence from investigations, including those of Larmon, that the cross-section of the intervertebral foramen is generally only 1 mm larger than that of the ganglion lying at its entrance. It is also known that the surroundings of the extradural root narrow like a funnel towards the foramen (what we have elsewhere called the "approach to the foramen", *i.e.*, the lateral recess of the spinal canal). Hence during the Lasègue, the thickest part of a root enters a smaller space and we are therefore very much inclined to believe that the sensation of pain is produced by the jamming of the slightly thicker root plus ganglion in this smaller space at the entrance of the foramen. The part played in this by a hernia is the changing of the root and the ganglion, which are irritated by the hernia and probably swell somewhat



With the information to hand from operations, it is barely conceivable, from the mechanical point of view, that a sharp pain irritant should arise from the shifting of a smooth-walled root sheath over a protuberance. It might be

Pressure-raising factors, like coughing, sneezing, straining, laughing, etc., may bring on or exacerbate radicular pain.

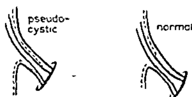
With hernia: (operatively verified)	
coughing	86%
sneezing	86%
straining	71%
(Krayenbuhl	90%)

The symptom is not pathognomonic, as it does not occur in 9% of verified hernias. Radicular pain was also initiated or exacerbated in patients without hernia ("negative explorations").

What is the source of this radiating pain? The view that the raised pressure causes a prolapsed disc to protrude further, thus compressing the root (Reischauer), certainly does not offer a comprehensive explanation, since the phenomenon also occurred without root compression, or without a disc lesion. It would be more correct, we think, to stress the *increased pressure in the dural sac*. This pressure can be raised in various ways (jugular pressure, head flexion symptom, rising from supine position, saline injection into the dural sac or root sheath) and the result is always about the same. We have verified this in selected cases of radiating monoradicular pain under such stresses. If the patient is made to strain during an operation, one can see the lumbar sac swelling up and the roots shifting slightly towards the foramina. It might therefore be that movement of the irritated, compressed or adhered root or cauda fibres is the source of pain. Against this, however, it is a fact that the phenomenon also occurred when root compression was not found at operation and the pathologist found no lesions of the cauda fibres upon anatomical examination after rhizotomy. It should be borne in mind that the symptom was presented pre-operatively and that during the exploration (under a local anaesthetic), after the root in question had been stimulated, the patient felt and reacted typically to the same pre-operative radiating pain, while we know of similar cases in which the pre-operative pain did not recur after the root had been cut. The records did not show whether in these cases an open communication existed between the dural sac and the root sheath.

It is our experience that *injecting fluid into an extradural root sheath* produces radicular pain The swelling goes down shortly afterwards. Assuming open communication with the dural sac, we expected the intradural fibres also to become numbed when Procaine was injected, and that proved to be so. In a very small minority of observed cases, the insensitivity was confined to the root concerned, yet it is a certain fact that the fluid ran out of the root sheath fairly quickly, as 2 to 5 c c. were injected into this limited space. Hence in these cases the fluid drained away distally (possibly, too, subdural-epiarachnoidally in a central direction) There must be a block somewhere between the intra-arachnoidal cerebrospinal space of the lumbar sac and the peripheral

root; otherwise, the fluid, standing under varying increased pressure, would likewise be able to drain off rapidly through the extradural root sheath distally. If, before performing a rhizotomy in the root sheath, one opens the sheath at about half a centimetre from the dural sac, usually fluid does not issue through the opening, but very occasionally it does! This points to the fact that the fluid block is not always situated at the same distance from the dural sac. In three cases – all three with pain in reaction to pressure-raising factors – we found a macroscopically detectable open communication reaching to 1½ cm from the lumbar sac (so-called cystic roots). In this the cauda fibres could be seen to fluctuate, while the cerebrospinal fluid was easily squeezed out of the root sheath, which filled up again immediately afterwards.



Von Lans, Feld and Tarlov have demonstrated arachnoidal occlusions in the beginning of the root sheath. Duus, injecting dyes, could not detect an open communication between the root sheath and dural sac; Lindblom found arachnoidal proliferations where the cauda fibres emerge from the dural sac (associated with very laterally situated hernias at a distance).



The foregoing facts, together, provide convincing evidence of the existence of a *mechanical* cerebrospinal fluid barrier. Its site, according to our experience, is somewhere between 0 to 15 mm from the point where the dural sac merges into the root sheath, but generally at 3 to 4 mm (We have not succeeded in detecting this barrier microscopically.) Hence there is fluid in a space of a few millimetres where the root sheath begins, in a bulge (or recess) of the arachnoid. It is possible to demonstrate this bulge by myelography with aqueous contrast medium. Raised pressure in the dural sac is transmitted caudally and swells the lumbar sac and the commencement of the root sheath, as has been seen with the naked eye. The arachnoidal bulge at the site must resist this or may yield to it. Under physiological conditions, raised pressure in this bulge does

not produce pain. If the symptom is positive, the general increased pressure in the lumbar sac does not produce a pain irritant in any of the other roots, but results in monoradicular pain. Hence the local relations must have changed. Either the arachnoidal barrier yields and extends over the fibres in the root sheath (the tearing of adhesions or proliferations cannot be involved, as the phenomenon may be repeated *ad infinitum*); in that case the bulge acts as a stenotic process and exerts pressure upon the root fibres. Or else the root itself has changed as the result of the local monoradicular process and is hypersensitive to the same increased pressure which caused no pain from the other roots, nor previously from this one.

Let us compare these suggestions with the conditions found in association with a hernia. It would then be conceivable that a bulging fold of arachnoid in the root sheath compressed by a hernia exerts additional pressure which exacerbates the same radiating pain (or initiates it) which had already been induced by the local pressure of the hernia. Root fibres already irritated will be all the more liable to succumb to the effect of this pressure.

If the symptom is positive without the complication of a hernia, one must be prepared to find irritable fibres, compression or some other kind of constriction or an abnormal extension of the arachnoidal bulge, or cystic root. On this hypothesis it is to be supposed that, even under physiological conditions, the arachnoidal fluid barrier at the commencement of a root sheath is plastic and subject to stretching under pressure. We can see no objection to this argument; on the contrary, findings with a "normal" root seem to corroborate it. It also helps us to picture how a cystic root sheath filled with cerebrospinal fluid comes into being.

It thus becomes clear that the reaction to pressure-raising factors cannot be a pathognomonic symptom, and, indeed, is not. For, we may expect it to be positive in association with:

Inflammation of the fibres.
Adhesion, coalescence with, or constriction of the root.
Oedema (?).
Compression.

The *head-flexion symptom*, associated with hernia, is positive in 31%. We call it positive if, on bending the head, radicular pain occurs in the regions where the patient usually feels it.

The patient can lie down, stand or bend forward, all preferably with the legs extended. If the pain should occur without straightened legs, it is a sign that the head flexion symptom is positive in a very marked degree.

The symptom is in the same category as the reaction to pressure-raising factors, has somewhat less value and is not pathognomonic. We do not think, as some do, that the radicular pain occurs through traction of a constricted root due to displacement of the spinal cord or the dural sac when the head is flexed. During cervical explorations we did not detect any appreciable displacement of the spinal cord upon maximum flexion (and extension) of the head in relation to the neck; this was photographed. Allowing for the parallax, the maximum displacement was certainly not more than 2 mm. This seems to us too little to exert distal traction upon an extradural root by transmission *via* the spinal cord and the descending cauda. The movement of the dural sac as we watched it was even less. Actually, the test can be so arranged as to leave no room for doubt. The patient in whom the phenomenon has already been found to be positive is made to lie down quite relaxed; the head is then moved gently in all directions so that the reassured patient notices that it does not hurt; finally, when the unresisting patient is lying peacefully breathing with his mouth open, his head is bent forward till the chin rests on the chest. The head flexion symptom has now become negative. Alternatively, the head can be kept firmly pressed down on the chest; the patient then becomes taut and holds his breath. When he has, of necessity, to take breath again, he is temporarily relieved of the pain thus caused.

We carry this head flexion test out by making the patient bend with the legs held straight. Just as the back shows signs of becoming rigid, we flex the head so that the chin comes to rest on the patient's chest. If this causes radiating pain, this pain will also occur when the patient coughs or strains in this position, even if radiating pain had not been experienced previously when coughing or straining in the erect posture.

Pain Signals during Neurological Examination

Local pain on pressure associated with hernia

Roughly	*At the level of*	*Number*	*Hernia at operation*
Lumbo-sacral	L.IV – L.V: 42%	(90) →	22 L.IV – L.V and 68 elsewhere
43%	L.V – S.I: 30%	(65) →	23 L.V – S.I and 42 elsewhere
	Elsewhere: 28%	(60)	
215		215	

Hence the location of the hernia found at L.IV – L.V in about 1 in 4 cases agreed with that of the pain felt on pressure, and at L.V – S.I in about 1 in 3 cases.

Lumbo-sacral sensitiveness to pressure is by no means pathognomonic for hernia and, indeed, is located by the patient more often than not at places where no hernia is found subsequently. We have moreover noticed that patients revealing no signs of a disc lesion at general examination were likewise sensitive to pressure in the lumbo-sacral region. Spurling, p. 52, however, is of opinion that a positive percussion test is almost pathognomonic of a ruptured intervertebral disc and sometimes makes it possible to differentiate accurately between lesions of the fourth and fifth disc.

Axial pain from pressure	3%
Peripheral points of pressure	21%
(Krayenbuhl	46%)
Pain from pressure upon superficial parts of the skleleton	?

A sensation of pain from pressure along the course of the sciatic nerve is generally regarded as a symptom of neuritis (pressure at the site of the buttock fold, or pinching in the Achilles tendo). This last test is, we think, reliable. It is sometimes described as "positive according to Abadie", but this is confusing because Abadie described the symptom in an entirely different context, *viz.*, in association with tabes.

The fact that the *sciatic nerve was sensitive* to pressure in 21% of verified cases of hernia is certainly instructive, because it implies that this symptom does not provide us with a datum for differential diagnosis between neuritis and an irritated root caused by a hernia nuclei pulposi. We do not know from what changes induced by a hernia the sensitivity to pressure of the peripheral nerve derives, but many think it is due to a secondary radiculo-neuritis caused by the hernia. The possibility of increased background activity by pressure on the nerve has to be borne in mind (see p. (501) (491) 362 (359)).

We have no knowledge of the incidence of pain from *pressure upon the superficial parts of the skeleton* We observed it in association with osteomalacia and osteoporosis, especially involutional osteoporosis. It is a symptom presented more often by women. The pelvic crest and the anterior surface of the tibia are the most sensitive, but apart form this, tenderness is indicated upon pressure on the sternum, the sacrum, the os pubis, the ankles and laterally on the upper arm – in between the muscles – on the humerus. Not infrequently, one finds that one and the same patient experiences pain from pressure on the places enumerated, but not, say, on the superficial parts of the skeleton of the under-arm, the ankles, etc. This tenderness upon pressure is due to irritation of the periosteum as the result of the bone process.

Anamnestic indications of pain

Walking	56%
Standing	45%
Sitting	50%
Recumbent	29%

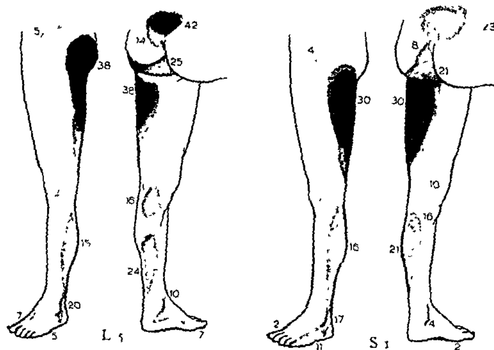
Spontaneous pain is observed largely in association with movements. Fortunately the number of patients suffering from pain when supine is small, relatively speaking; otherwise conservative treatment by prescribed bed rest would be impossible.

Rising	58%
Pain on beginning to move after rest ("Loosening up" symptom)	23%

These are symptoms presented in a fairly typical manner by the patient. They point to spondylarthrotic components occurring either independently or combined with another disorder.

Radiating pain
86%

	Coccygeal region	*Ilio-sacral region*	*Buttock*	*Groin*	*Thigh hip*	*Knee hollow*	*Calf*	*Lat. lower leg*	*Med. ankle*	*Lat. ankle*	*Instep, big toe*	*Lat. instep, little toe*
Hernia *at L.5* in %	42	14	25	5	38	16	24	15	10	20	7	5
Hernia *at S.1* in %	23	8	21	4	30	10	21	16	4	17	2	11



This shows that there is no significant difference between the radiating pain produced by a lesion of the L.5 root and that following a lesion of the S.1 root. For the same opinion see Spurling (1958) p. 46.

We see from this table that radiating pain is felt far more often in the upper leg than the lower. More numerous still are the patients who complain of pain radiating from the back laterally towards the lumbar and coccygeal regions, though it remains to be seen whether or not this is to be counted as radiating pain. This makes it difficult to establish typical regions of radiating pain conformable with the "known" patterns of innervation of the roots; in the lower leg these areas seem more clearly differentiated side by side (big toe, little toe).

The number of statistics showing patients constantly indicating pain radiating to the big toe and the little toe is truly amazing in the light of our experience of clinical histories, in which the numbers were small. Therefore, to find the innervation pattern of a given root, more reliable evidence will be the radiation associated with mechanical stimulation of just one root to be tested in a patient under a local anaesthetic.

In some of the cases verified at operation, radiating pain did occur preoperatively, while in others it did not:

Typical hernia (explored on account of paresis and other neurological symptoms) Negative exploration for disc lesions	Radicular pain, or No radicular pain
Spondylarthrosis (detected radiographically)	Radicular pain, or No radicular pain. Back complaints only, or No complaints.
Radico-neuritis	With and without pain in back or legs
Manifestations of degenerative back without hernia	With and without pain in back or legs.

Conclusion

On these grounds we may conclude that subjective indication of radiating pain does not provide reliable evidence for diagnosis or locating. Bradford and Spurling (p. 106) have the following to say on the matter: "Localized pain without paresthesia or hypesthesia is not very reliable in identifying the roots involved".

As a symptom, the radiation of pain has no pathognomonic value for the aetiology of the malady. Depending as it does upon the subjective indications of the patient, it is an unreliable datum for differential diagnosis between organic and psychogenic complaints. This notwithstanding, the examiner will,

naturally, question the patient in this sense. Like so many other practitioners, we are so accustomed to this unreliability that we have ceased being surprised to find that the patient's statements do not tally with the neurological picture or other available evidence. The chief value of this part of the clinical history is that it provides an impression of the severity of the complaints and of the patient's psyche; and it is a useful impression, to our mind, because it helps the physician to formulate a diagnosis of probabilities as regards back complaints. The findings at neurological examination can do no more than underline such a diagnosis; they cannot provide certainty, as will appear from the following.

By *radiating pain* we mean a sensation of pain something like an electric current running from the back downwards, for example. When it occurs, the patient feels as though the pain were travelling through the buttock, say, the upper part of the leg (missing the knee and the hollow of the knee) down into the calf and, sometimes, into the foot and toes. On the other hand, if the patient states that the pain seems to rise upward from the calf, perhaps, into the hip or higher still, we do not call this "radiating" pain. Causalgic sensations are also liable to rise upwards, but they are so different in character that they could hardly be mistaken for the "radiating pain" we are considering.

Sensations of pain can be classified as follows:

I Severe, stabbing pain (as though inflicted with a knife or drill, whether radicular or not in character).
II Sensory disturbances accompanied by pain (hypaesthesia dolorosa; usually a burning sensation).
III Paraesthesia with pain, *i e*, a feeling of numbness, "sleeping", tingling, itching, accompanied by a burning, causalgic sensation of pain or of a painful electric current.
IV Causalgia
V Phantom pain

Paraesthesia 37% (associated with hernia)

These sensations, the description of which depends on the patient's personal judgment, are often vague. The patient suffers loss of sensation (which can be verified objectively), experienced sometimes like a strange, *numb sensation*, and is generally unaware that his sensory acuity has diminished. A *tingling* sensation may ramify in a monoradicular area. These patients are prone to feel as though a leg "had gone to sleep" as soon as a peripheral nerve is squeezed, if only for an instant. In addition, the patient sometimes mentions a "heavy, tired feeling" in the legs which, he says, seem to be powerless. This is sometimes referred to in the literature as the "syndrôme d'effort". It is not clear whether these complaints are manifestations of paraesthesia.

Occasionally, indeterminate, spastic pain is felt – seeming to be more like paraesthesia than true pain – in association with peripheral arteriosclerosis and also with arthrosis of the hip joint. It might be designated as "ascending paraesthesia". It is often difficult to differentiate between this and intermittent claudication and allied abnormalities, producing cramp in one calf or both, which is brought on by exertion and relieved by rest. Similar to it is the dysbasia-like cramp associated with the syndrome of the stenotic canal according to Verbiest, an almost pathognomonic feature of which is that, together with the "syndrôme d'effort", it soon disappears if the patient lies down.

It is our impression that, rather than being localised segmentally, paraesthesia occurs approximately in the same innervation patterns as prevail when the sensation of pain is felt in similar cases and, in other cases, when objectively verifiable sensory disturbances exist.

Sensory Disturbances

Sensory disturbances. These occurred in 41% of the operatively verified hernia cases. But they also occurred with the so-called negative explorations in association with backs showing signs of degeneration without hernia and also accompanied spondylarthrosis, with and without radicular pain. According to Aitken and Bradford (Waris, p. 45), sensory disturbances were associated with 44% (27 of 62 cases) of negative exploration for hernia nuclei pulposi. The occurrence of these disturbances belongs to the clinical picture of peripheral neuritis, but they are then varied by superficial hypersensitivity in innervation patterns not limited to one root.

If a sensory disturbance is to yield useful information for diagnosis or location, it is important to ascertain whether it is monoradicular, in the innervation area of which root it occurs and whether it is compatible with a root lesion, if such should be found subsequently.

We have to distinguish between surface-sensations and depth-sensations. A good example is the sensory innervation of the inguinal region, radiating pain from the groins is almost typical for a hernia at L III – L IV or L.II – L.III, corresponding to a lesion of the L.4 or L 3 root. In such cases there is no detectable disturbance of sensation in the skin of the ingual region, nor does the patient complain of a numb feeling there It is as though the pain radiates underneath the groins, sometimes to the external reproductive organs, the pain seems to be seated at a deeper level, some way down in the groin, the abdomen or where the abdomen merges into the thick part of the thigh The pain disappears when a hernia at a corresponding level, involving the L 4 or L 3 root, has been removed

The bundles of nerves serving the surface-sensibility in this area, on the other hand, pass through the L 2 (and L 1) roots (nervi ilio-inguinales) This was demonstrated during rhizotomy of this (these) root(s), such as is performed for stubborn

neuralgia paraesthetica. Thus, in the same region, surface-sensibility is fed by different roots from those serving depth-sensibility, which is an important point to remember when interpreting these complaints in an attempt to locate a lesion, if any.

We shall first discuss surface-sensibility as one imagines this to be projected in dermatomes.

We speak of an *S.1 syndrome* in medical practice when, due to a lesion of this root, the Achilles tendo reflex is negative or reduced, sensibility along the skin of the lateral shank, the lateral edge of the foot and the little toe is disturbed, with pain and paraesthesia occurring in this region. If the knee reflex is reduced or negative and disturbed sensation, pain or paraesthesia occurs in the hip, instep and big toe, we call this an *L.5 syndrome*. This is where our difficulties begin, because the majority of investigators have it that the reflex tract for the knee tendon follows a route *via* the L.4 or L.3 root and for this reason deny that the reduced reflex belongs to the L.5 syndrome.

Radiation of pain following root irritation

According to pre-operative syndromes verified at laminectomies,		As per Foerster (p. 292)
	L.1	Groin
Skin of lateral thigh and groin	L.2	Anterior thigh
	L.3	Knee
Deep to the hip and groin	L.4	Inside ankle, big toe
Instep, big toe	L.5	Instep, all toes
Outside ankle, lateral edge of foot, little toe	S.1	Sole, heel
Posterior thigh, hollow of knee, calf	S.2	Posterior thigh, hollow of knee
Gluteal fold	S.3	Gluteal fold
	S.4 S.5	Anus, penis-vulva

We collected the monoradicular disorders of L.5 and S.1 from 500 laminectomies, then selected 100 cases of each group in which, according to the records, the lesion was situated exactly under one root (*e.g.* a bud hernia). We eliminated cases of multiple abnormalities, positive myelographs at two levels as well as atrophies and diminution of power, to avoid double syndromes. Though one can never be sure that there is no additional factor, such as the lesion of two roots caused by one hernia at the same level, we thus approximated the necessary conditions fairly closely. The groups were larger at first, but in the course of our work many cases were dropped which did not present a (clearly) radicular syndrome pre-operatively. The pre-operative (neurological) findings were classed under these groups and we record only the following from among the many facts collected:

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anatomical variation. In other words, the L.5 root runs under the 24th vertebra (L.V)".

Lewin, p. 759.

Falconer, quoted by Lewin: "Sensory impairment in the thigh and buttock, in a patient with sciatica, can no longer be regarded as evidence that *several* roots of the cauda equina are involved or that the patient is *hysterical because his sensory pattern does not conform to the classical dermatome charts*".

Droogleever Fortuyn, *Brain* 72 (1949) 568.

"It may be noticed that in most patients with involvement of lower lumbar and upper sacral roots more than one area is found".

"If these bands (areas of Keegan's scheme) represent dermatomes this would imply quite a revolution in neurology".

Armstrong, pp. 84, 95, 194.

"It is true that the areas described are *not exact* and that some overlap occurs".

Explanation: (pp. 95 and 194) Variations through abnormal lumbo-sacral plexus and/or variations in the number of mobile vertebrae?

Armstrong, p. 92.

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"Because of the possibility of wide overlap and considerable *variation* ... even the most accurate plotting ... cannot necessarily supply irrefutable evidence of the level of a lumbar disc lesion".

Love, *Acta Orthopaed. scand.* 18 (1949) 142.

".. emphasizes the great variations in the construction of the lumbo-sacral plexus and stresses that the level of space-occupying lesions in this region *cannot be correctly* diagnosed by neurological examination alone".

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One third of the patients do not present any neural loss symptoms . . ."

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Referring to his findings in the study of dissected material, he predicted in 1910 that "local diagnosis on the basis of functional disorders cannot be other than approximate".

Spurling, 1958, p 58.

"In well over half of all cases variations in the patterns of dermatomes .. make the results (of sensory tests) confusing rather than helpful"

Bradford and Spurling, p 106

"Localized pain without paresthesia or hypesthesia is *not very reliable* in identifying the roots involved".

p. 51.

"Keegan published dermatomes for the lumbar and sacral nerves which have *not corresponded* to our findings or to the findings of others".

Disturbed sensation:

	Total
in 500 hernias	41%
in 100 selected L V – S.I hernias	36%
in 100 selected L.IV – L.V hernias	46%

We thus gained some insight into the peripheral fields of projection of disturbed sensation resulting from 36 purely monoradicular S.1 (and 46 L.5) lesions.

	Little toe lateral edge of foot	*Lateral shank*	*Hip Big toe*	*Elsewhere*
S 1 lesion	54%	27%	—	19%
L.5 lesion	30%	40%	15%	15%

This shows that the sensory innervation patterns of the L.5 and S.1 roots are not as specific as the confident interpretation of them in practice would suggest.

Davis, Martin and Goldstein likewise compared the pre-operative syndrome with the level of the lesion in 500 hernia cases verified at operation. They found so much variety that they came to the following conclusion: "Sensory changes in 500 patients with herniated nucleus pulposus showed no correlation between the sensory pattern and the level of the lesion" (p. 138). (This investigation was not completed by root irritation during operation.)

We found the following comments in the works of other authors whom we quote in support of our argument.

Davis and co-workers, *Neurosurg.* 9 (52) 133	"*No correlation* between sensory disturbances and level of the lesion"
Rousseaux and co-workers, *Rev. neurol* 5(52) 530.	"*Incongruity* between clinical findings and location".
Kostic and co-workers, *U.I M.C* 1 *I* p 73	"Changes in superficial sensibility are *neither regular nor strictly limited* to the individual dermatomes".
Lewin, p 756	"The neurological signs are *not* always *conclusive* as to localisation partly because of *variation* in dermatome distribution"
Lewin, p. 758	"The exact anatomical significance of dermatome areas is *somewhat uncertain*".
Lewin, p. 757	" dermatome chart is *helpful* in neurologic diagnosis and anatomic teaching"
Lewin, p. 759	Keegan, quoted by Lewin In cases of transitional vertebrae the relation between vertebral level and cauda root remains unchanged if allowance is made for the displacement of the vertebra due to the

anatomical variation. In other words, the L.5 root runs under the 24th vertebra (L.V)".

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Without mentioning variations, the authors have the sub-heading "*Approximate dermatome*" above their charts.

Reischauer, p. 34.

"Die *Konstanz* der Dermatome ist erstaunlich grosz, die subjektive Lokalisation von Schmerz – oder Gefuhlsstorungen . . . uberraschend bestimmt. Wo Sensibilitatsstorungen fehlen hat das Schmerzband den gleichen lokalisatorischen Wert".
(In holding this opinion, Reischauer stands practically alone among numerous authors writing in no uncertain terms of the variability and unreliability of [illegible]

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Roeder, p. 8.

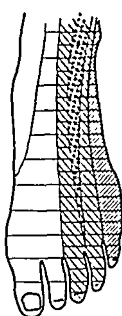
"Two-thirds of the hernias verified at operation cause sensory disturbances".
"Die operativ verifizierten Bandscheibenvorfälle zeigen, wie gross die *Konstanz* dieser Dermatome ist".

According to some authors, the sensory innervation of the big toe is supplied by the L.4, L.5 and/or S.1 roots:

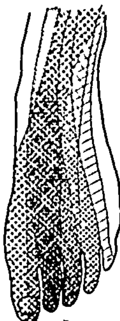
Root L.4 → big toe. Edinger, Bare, Biemond (Fig. 109b), Keegan.
Root L.5 → big toe. Armstrong, Bradford and Spurling, Falconer, Krayenbuhl, Norlén (→ Lewin), Reischauer, Webb.
Root L.5 → big toe (S 1) Richter (→ Krayenbuhl, p 28) (as variant)

Upon comparing the charts drawn up by various investigators respecting sensory disturbances in the course of the peripheral roots from the cauda equina, they might seem, roughly speaking, to agree fairly well, but on closer inspection and on testing the charts against actual findings, it will be found in the case of one particular individual that 3 or 4 of the 4 or 5 charts do not correlate with the facts. Keegan's chart is little else than a working plan, as the innervating distribution it gives is too good to be true. Compared with Bolk's charts, both show straight borderlines between the dermatomes, whereas the borders found by Foerster's method run erratically and overlap. Furthermore,

we projected several charts (Armstrong, Barr, Bolk, Foerster, Keegan, Krayenbühl, Webb, Johnson) in one drawing, when it transpired that almost the whole of the lower part of the leg or the foot is occupied by the innervation area of one root.



S 1



L 5

Therefore, with the foregoing in mind, we have reason to suppose that variations occur in the sensory innervation patterns.

We complemented our enquiry by monoradicular *mechanical* irritation of roots in patients under a local anaesthetic during operation. The number of tests performed, however, was too small to be expressed statistically. This was because, the moment typical pre-operative pain was signalled or a prominence was clearly felt while nibbling the arch or during transdural palpation, the intradural roots were anaesthetised with Procaine and no further indications of pain or sensory disturbance could therefore be obtained. We feel that we are bound by our duty to the patient to do this, but on several occasions we were able to depart from this rule. We were able to ascertain (some herniatomies, negative explorations, rhizotomies, etc.) that monoradicular irritation of the L.5 and S.1 roots usually caused the known distribution of radiating pain, roughly to the hip and big toe, and the lateral aspect of the shank, the lateral edge of the foot and the little toe, respectively. Yet quite a few patients – we estimate about 20% of them – were quite unable to distinguish irritation of L 5 from that of S 1 (!). Undoubtedly many surgeons will have been faced

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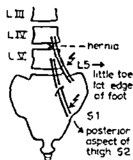
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Root L 4 → big toe:	Edinger, Bare, Biemond (Fig. 109b), Keegan.
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Operatively: hernia nuclei pulposi L.V – S.I.
Monoradicular irritation of S.1:
pain → *hip, big toe* (clinically L.5)
pain → lateral edge of foot (clinically S.1).
The hernia nuclei pulposi might have caused the pre-operative double syndrome through lesion of two roots at one level. The result of the monoradicular irritation of the anatomical S.1 root during the operation suggests that the L.5 fibres run via S 1 to the big toe.

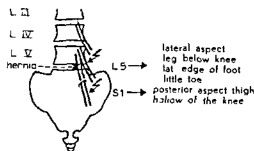
Normal spine (7 C + 12 Th + 5 L) Pat. (♂) H. 14257



Pre-operatively:
S.1 syndrome: { pain in lat. edge of foot and little toe. { Reflexes: A.t.: negative.
Myelographically: Hernia nucl. pulp. L.IV – L.V.
Operatively: Hernia nuclei pulp. L.IV – L.V.
Monoradicular irritation of the anatomical root L.5:
typical pre-operative pain → lateral *edge of foot and little toe* (S.1).
Monoradicular irritation of the anatomical root S.1:
No pain in lateral edge of foot or little toe, but
Pain → *posterior aspect of thigh* (S.2).

Note Macroscopically the aspect of the anatomical S.1 root was normal (colour, position, girth)
L.5 was irritated at the site of the hernia, hence it could not have been mistaken for S 1 *A post-operative check-up confirmed the localisation.* (Silver clip in disc L.IV – L.V)

Normal spine (7 C + 12 Th + 5 L) Pat (♀) v. d. V. 23257

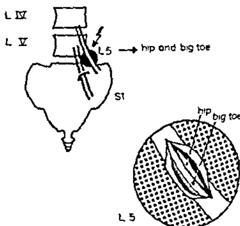


Pre-operatively:
Pain in posterior aspect thigh, hollow of knee (S.2 syndrome).
Sensibility intact.
Reflexes: Nothing unusual.
Myelographically: Hernia nuclei pulposi, dubiously L.V – S.I.
Operatively: Prolapse L.V – S.I.

Monoradicular irritation of the anatomical S.1 root:
Typical pre-operative pain → *posterior thigh* and *hollow of the knee* (clinically S.2).
Monoradicular irritation of the anatomical L.5 root:
Radiation of pain → lateral shank, *lateral edge of foot and little toe* (clinically S.1!)

with this awkward situation some time or other; it occurs when one is thwarted from relieving the patient of his radicular pain by a rhizotomy because he is unable to intimate any typical radiation of his pre-operative pain. We admit in all fairness to having been irked by this sometimes; all too readily one suspects the patient of not paying proper attention, of not co-operating or of being ridden by psychogenic complaints. But we are persuaded that there are some people who really cannot differentiate upon irritation of L.5 and S.1 and that this is chiefly due to the composition of the roots themselves.

Let it suffice to mention a few particulars found in a "normally" constructed spinal column. This patient was able to distinguish between irritation of L.5 (hip-big toe) and of S.1 (lateral edge of foot, little toe). Moreover, during selective sensory rhizotomy in the root sheath of L.5, he intimated pain *separately* radiating to the hip when the uppermost sensory fibres of the root were stimulated, and pain *separately* radiating to the big toe when the lower sensory fibres of the same root were irritated. This might provide evidence of an arrangement of fibres in a caudal nerve root itself in a certain order corresponding to the areas to which they run.

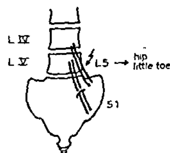


In a normal vertebral column there existed pre-operatively a clinical S.1 syndrome (case, not further particularised, taken from notes on earlier observations).

Hernia nuclei pulposi L.IV – L V under the L.5 root confirmed myelographically and at operation

Upon *mono*radicular irritation of L 5 during the operation the pain radiated to the *little toe*

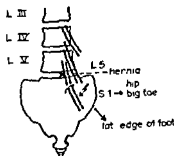
This finding makes us wonder whether in this case fibres passed through L.5 to the little toe (= S 1 innervation area)



Normal spine
(7 C + 12 Th + 5 L)
Pat. (♀) S 16257
Pre-operatively·

L.5 syndrome	pain in hip and big toe, sensory disturbance in hip.
S.1 syndrome	pain in lat edge of foot, sensory disturbances in lat edge of foot and little toe
Reflexes:	A. t. negative

Myelographically: hernia nuclei pulposi L V – S 1.



certain lumbar characteristics (lumbarisation) or L.V presents some features of the sacral type (sacralisation). If the word "sacralisation" is used erroneously to denote a lumbarising tendency of the twenty-fifth vertebra (S.I), it implies that this S.I is considered to be a dropped lumbar vertebra L.V with all its consequences (l). The root passing under it would then, again erroneously, have to be called the L.5 root. This would involve departure from the usual neurological innervation patterns; in tests for a suspected L.5 lesion a negative Achilles tendo reflex, for example, would be found and the examiner would assume, wrongly, that there was a variation in reflex. In doubtful cases this may affect the diagnosis and obscure the indications.

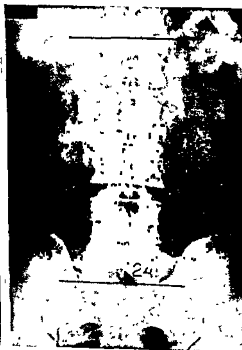
Mistakes of this kind are by no means imaginary. Schmorl's statistics show the incidence of transitional vertebrae as varying from 0.6% to 26%. Kühne and co-workers found that gross anatomical transitional vertebrae occur in one third of the human race, but taking minor anatomical details (transitional tendency) into account, they found the following:

Normal conditions prevail in only	7%
Sacralisation (tendency)	34%
Lumbarisation (tendency)	59%

If only the neurological variations could be shown convincingly to stand in relation to the transitional vertebrae, the incidence of the latter is undoubtedly



Lumbarisation



Pseudo-lumbarisation

Normal spine (7 C + 12 Th + 5 L) Pat. (♂) L. 28257

Pre-operatively:



L-4 syndrome

Pain: In tibia and groin.
Disturbed sensation: In anterior aspect shank and lateral aspect of calf.
Reflexes: A. t., left lower than right. K. t., left lower than right

Myelographically: Hernia nuclei pulposi L.IV – L V.
Operatively: Hernia nuclei pulposi L.IV – L.V.
Monoradicular irritation of the anatomical L.5 root
Radiation of pain → *tibia and groin* (clinically L.4).

We may infer from the foregoing that *variations* occur in the clinical-neurological innervation patterns. We also see that the fibres may be arranged in a certain order in the cauda equina roots; it was observed that the clinically verifiable function of one root takes place, entirely or partially, in another root, is (temporarily?) performed by the latter or wholly transferred to it.

To make use of these innervation patterns for diagnostic localisation is tantamount to calculating chances, since mere guessing between L.IV – L.V and L.V – S.I gives the correct localisation in about 50%; the interpretation of the neurological picture increases the chances by a mere 20%.

We may now ask whether diagnostic localisation – and with it the operative prognosis – could be improved if the circumstances making the interpretation of neurological symptoms for localisation unreliable were better understood. We have considered whether the *phenomenon of transitional vertebrae* would be enlightening, whether, that is, these vertebral anomalies are perhaps responsible for the variations in the innervation patterns. This phenomenon is the cause of many misunderstandings on account of the nomenclature. We recommend constructing a chart for every patient with transitional vertebrae and including notes of the facts revealed by radiological examination of the whole spinal column. We nearly always did this in the cases we shall mention presently.

The term "lumbo-sacral transitional vertebra" expresses variation in the transition from the lumbar character of the 24th vertebra (L.V) to the sacral character of the 25th vertebra (S.I), (or, mutatis mutandis, from the 25th to the 24th vertebra). It is necessary to distinguish between the true, material transition in which there are either only 23 vertebrae above the sacrum (sacralisation) or 25 (lumbarisation), and the pseudo-transitional vertebra. The varying form of the latter is only the manifestation of a transitional tendency; actually there are the normal number (24) of vertebrae above the sacrum, but S.I has

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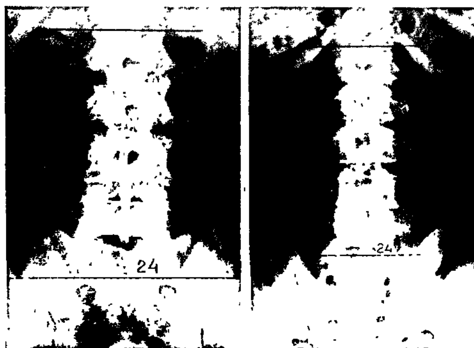
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Lumbarisation



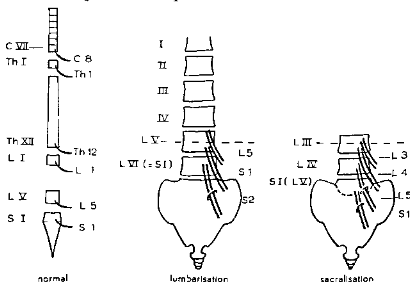
Pseudo-lumbarisation



Sacralisation Pseudo-sacralisation

sufficiently frequent to place that relationship, maybe, in a causal context

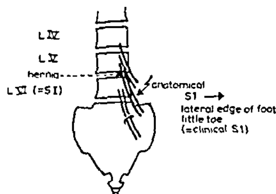
The important point to decide now is the course followed by the roots and fibres of the cauda equina with respect to a transitional vertebra. In a normally



constructed spinal column, the root emerges from under a given thoracic and lumbar vertebral body, after which it is named. Thus, under the L.V arch the L.5 root runs through the L.V – S.I foramen and, under the S.I arch, the S.1 root passes through the S.I intervertebral canal towards the first sacral foramen between S.I and S.II. In doing so, the L.5 root passes the L.IV – L.V disc and perhaps laterally the L.V – S.I disc; the S.1 root, on the other hand, runs only over the L.V – S.I disc (disregarding 11% variations, according to Thurel; see p. 198).

In current opinion, as also expressed in the literature (*e.g.*, Lewin, p. 758), these relations are maintained in the presence of transitional vertebrae. This, we find, is correct, provided the nomenclature of the caudal roots be correlated with the shifted nomenclature of the vertebrae. For example: by and large, a clinical (neurological) S.1 innervation pattern remains valid for an anatomical S.1 root even in the presence of a transitional vertebra; hence the function appropriate to the anatomy is maintained.

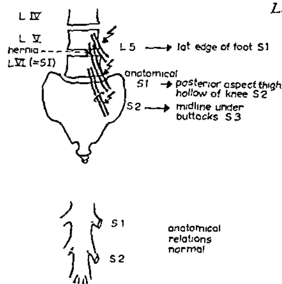
Transitional spine: Lumbarisation.
(7 C + 12 Th + (*5* + *1*) *L*)
Pat. (♂) D. 14756.



Clinical history (For several years S.2 syndrome: pain → posterior aspect thigh, medial edge of foot).
Recurrency. Former pain ceased; now different pain → lateral edge of foot and lat aspect shank.
S.1 syndrome: Disturbed sensation, lateral edge of foot and little toe.
A. t reflex: negative.
Myelographically: *L IV* – *L VI* (L.IV – L.V) dubious.
Operatively On account of S.2 syndrome in clinical history and existing S.1 syndrome, exploration at L V – L VI and L.VI – S.I. Hernia nuclei pulposi L.V – VI and old, small, ossified hernia nuclei pulposi L VI – S.I
Monoradicular irritation of the anatomical *S.1* root Typical pre-operative pain lateral edge of foot and little toe (clinically *S 1*).
One year after operation: Pre-operative pain ceased, negative A. t. reflex became low positive, no disturbance of sensation in lateral edge of foot and little toe; but functional listhesis L.IV in relation to L.V.
L 5 syndrome. "recurrent" pain. big toe, hip, knee reflex reduced to negative.

This patient suffered from a disc lesion or hernia nuclei pulposi of L.VI – S.I, L V – L VI successively and now probably of L.IV – L.V. We established by irritation during the operation that, despite the lumbarisation, the clinical function of S.1 corresponded to the anatomical location of S.1.

Thus we were able to demonstrate the normal relation between cauda and true lumbar transitional vertebrae by mechanical monoradicular irritation of the root. Exceptions do occur, *viz.*,



Lumbarisation (7 C + 12 Th + (5 + 1) L.
Pat. (♂) H. 19257.

Pre-operatively:

S.2 syndrome { Pain: Posterior aspect thigh, knee, calf, ankle. Sensory disturbance: lateral edge of foot.

Operatively: Hernia nuclei pulposi L.V – L.VI

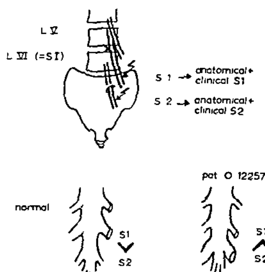
Monoradicular irritation of the anatomical *S.1* root:

Typical pre-operative pain → posterior aspect thigh and knee (*S.2*).

Monoradicular irritation of anatomical *S 2* root:

Pain → middle line under buttock (*S.3*).

Monoradicular irritation of anatomical L 5 root: Pain → lateral edge of foot (*S.1*). These particulars would seem to show that in this case the cauda underwent *greater cranial displacement than the spinal column*



Lumbarisation (7 C + 12 Th + (5 + 1) L.
Pat. (♂) O. 12257.

Pre-operatively:

S 1 syndrome { Pain. lateral shank. Sensory disturbance: shank and edge of foot. A t. reflex. negative

Myelographically: L.V – L.VI.

Operatively: Hernia nuclei pulposi L V – L VI

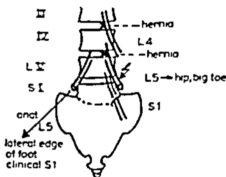
Monoradicular irritation of the anatomical S.1 root: Pain in clinical S.1.

Monoradicular irritation of the anatomical *S.2* root: Pain → posterior aspect thigh and knee (clinically S.2), *midline under buttock (clinically S.2?)*

Upon exploration, this anatomical S.2 root (at L.VI – S.I) proved to be far thicker than is normally the case, certainly in proportion to the S.1 root at a higher level.

Why, we may ask, should this S.2 have been thus enlarged? The radiation of pain to the midline of the buttock suggests that maybe this thick S.2 contained fibres for S.3.

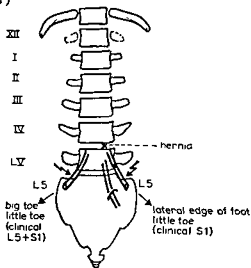
Pseudo-sacralisation (7 C + 12 Th + 5 L).
as the result of
lumbarisation tendency S.I.
Pat. (♂) v. W. 16257.



Pre-operatively. Pain: Hip, big toe.
L.5 syndrome. Sensory disturbances: Hip and lat. aspect shank.
(L.4 syndrome?): Reduced knee reflex.
Reduced motility foot.
Myleographically. L.IV - L.V (L.III - L.IV dubious).
Operatively. Hernia nuclei pulposi L.IV - L.V - L.III - L.IV.
Monoradicular irritation of anatomical L.5 root:
Homolateral. like pre-operative complaints → big toe, hip (clinically L.5)
Contralateral → lateral edge of foot (clinically S.1!).
Note. Four years previously patient had contralateral pain in the lateral aspect of the shank and the edge of the foot!!

The different intimations as to radiation of pain upon irritation during operation suggest that in this pseudo-sacralisation the L.5 roots were laid down asymmetrically. Probably the contralateral L 5 root contained fibres of S.1.

Pseudo-lumbarisation (*sive lumbarisation Th. XII*)
(7 C + (12 — 1) Th + (5 + 1) L.)
Pat. (♂) H. 25257.
(Surgeon. Dr. A C de Vet)



Pre-operatively:

S 2 syndrome: Pain Posterior buttock, thigh, hollow of knee and calf. No sensory disturbances. No reflex abnormalities.

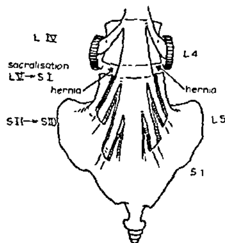
Myelographically L IV - L.V
Operatively. Hernia nuclei pulposi L IV - L.V
Monoradicular irritation of the anatomical L 5 root
Homolateral Pain → lateral edge of foot and little toe
Contralateral. Pain → *first big toe*, then little toe after all (clinically S.1).

Thus, in association with this pseudo-lumbarisation, fibres functioning like those of S.1 ran through one of the anatomical L.5 roots. The same condition probably obtained on the other side, though there was evidence of some fibres functioning like L.5 there as well

Davis's material contains particulars of 16 transitional vertebrae, all of which were lumbarisations.

Pre-operatively:	*Operatively:*
9 clinical S.1;	Hernia nuclei pulposi L.V – L.VI = anatomical S.1
1 clinical S.2;	Hernia nuclei pulposi L.VI – S.I = anatomical S.2
1 clinical S.1;	Hernia nuclei pulposi L.VI – S.I = *anatomical S.2*

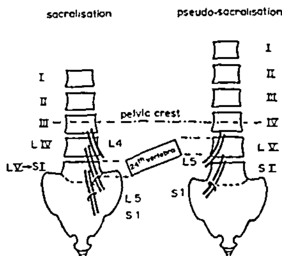
These findings correlate roughly with ours, but they were not verified by monoradicular irritation during operation. No explanation is suggested.



Whereas lumbarisation occurs abundantly in our material, *true* sacralisation is exceedingly rare. When we had arranged the cases in groups, we found that several "sacralisations" were no more than a manifestation of a lumbarising tendency. The fact that the incidence of sacralisation is half as frequent as lumbarisation primarily accounts for the rare occurrence of true sacralisation in our operation statistics. Moreover, when there is genuine sacralisation, the anatomical L.5 as well as the anatomical S.1 roots pass through rigid uninterrupted bony canals. The L.4 roots, in the incisura inferius of the L.IV arch, pass through the L.IV – L.V intervertebral foramen (which is, moreover, wide) at a high level and are therefore not much in contact with the L.IV – L.V intervertebral disc. Hence it is only the proximal part of the L.5 roots that, in the event of genuine sacralisation, run the risk of being involved in a hernia (from the L.IV – L.V disc). A surgical radicular case as the result of a hernia nuclei pulposi is therefore less likely to arise (no S.1, little or no L.4, less L.5) and this is why true sacralisations come less frequently into the neurosurgeon's hands.

Latterly we have not had a single case of genuine sacralisation qualifying for surgical intervention and have therefore been unable to check up the innervation patterns by irritation during operation. Since radiological examina-

tion of the whole spine used not to be performed as a general rule, there was no purpose in investigating correlations between clinical data and findings at operation in the past. Judging by our recent experience, we are very much inclined to believe that several, if not many, of the transitional vertebral columns entered as "sacralised" in early records were in fact lumbarised forms of the first sacral vertebra.



With *true* sacralisation, the course of the roots of the cauda equina is, theoretically, as follows: The anatomical *L.5* root runs under the sacralised transitional vertebra L.V, which became "vertebra S.I", and emerges from the topmost opening in the sacrum. In *pseudo*-sacralisation (lumbarising tendency), it is the *S.1* root that runs through this opening. We are very curious to know what the results will be when we are able to trace this "missing link" in our series at operation, by irritating the roots.

Meanwhile, we have come across in Norlén (p 46) the description of a true sacralisation (4 lumbar vertebrae above the sacrum) By mechanical monoradicular stimulation during the operation, he discovered that the anatomical root S.2 (which, taking the sacralisation into account, is, therefore, really S.1) functioned clinically in the manner of the S 1 root.

We have shown in a number of true lumbarisations that the relation between the cauda and the spinal column is not disrupted. Broadly speaking, from the clinical aspect, the function of the cauda roots shifts in proportion to the anatomical shift, while this is found to have shifted proportionately to the shift of the spinal column.

We also know that the neurological innervation patterns in "normal" and pseudo-transitional vertebral columns vary a great deal. It was found that pain similar to that occurring in the pre-operative syndrome was initiated by the mechanical monoradicular irritation of an injured root at the level of the abnormality detected. The neurological variations associated with the pseudo-transitional spine indicated a tendency to asymmetry.

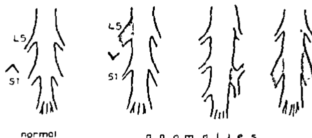
According to Kühne, overt and subclinical manifestations of transitional tendencies occur in 93% of human spinal columns. We include among these: pseudo-lumbarisation, sacralisation of the vertebral canal, exceptionally wide vertebral canal, deformity of large L.V lateral process, lumbar ribs and pseudo-spina bifida occulta of S.I – S.II. All these are cases representing disorganised morphology of the vertebral column, of the vertebra and/or of the vertebral canal.

We observed an incongruity between the pre-operative neurological syndrome and the level of the abnormality found

in 11% of 50 normal spinal columns and
in 36% of 100 pseudo-transitional spinal columns.

Furthermore, it is our experience that root anomalies frequently occur in conjunction with anomalies of the spinal column. It is highly probable that there is a causal connection, but we could not prove it with significant statistics. *We postulate the existence of some relation between variations in the neurological innervation patterns, root anomalies and anomalies in the morphology of the vertebrae.*

Let us clarify this by considering the following observations. By no means rarely a root is found to be very much thicker at the site than is compatible with the known anatomical relations (Hanraets, 1953). Root L.5, which is generally thinner than S.1, may be the thicker of the two; S.1 can be amazingly thick if it runs side by side with an unusually thin root, while the neighbour of an excessively thick root may be lacking altogether.



What could cause this *distension of a root?* Thurel ascribes root pain in association with hernia nuclei pulposi to *oedema* of the roots. The occurrence of such

a degree of oedema has yet to be proved. And at all events, this possibility can be disregarded, because thickened roots are also found unassociated with hernia.





Radiculitis

Anomaly; microscopically: no oedema, no radiculitis nor ectopic ganglion

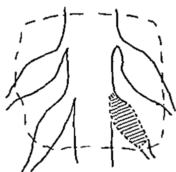
The next suggestion is *radiculitis*. If there is any proffered explanation that we reject out of hand it is that all these distensions are signs of inflammation of the root. No-one who has once seen and palpated a red, thickly swollen and exceedingly painful inflamed root will ever be mistaken again. The distended root, as an anomaly, is of normal whitish-yellow colour, displays no pathological vascular marking in the root sheath and is not excessively painful when touched. Having seen radiculitis in other cases with our own eyes, it is far from our intention to deny its existence. To get an idea of the incidence and anatomical proofs of non-specific radiculitis as the cause of radicular pain, one should refer to investigators who are convinced of this causality. A monograph by Krischek appeared in 1956 under the title "Neuritis oder Bandscheibe". Although it is the writer's intention to explain many, if not all, radicular syndromes on the assumption of a radiculo-neuritis, he was

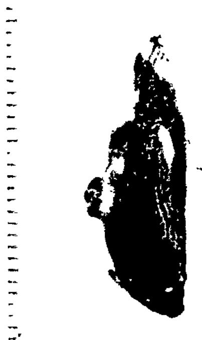
able to collect only a few cases from the world's literature in which the inflammation was detected by the pathologist (one plexus neuritis, some radiculitides associated with chronic rheumatism and four times signs of inflammation in resected roots in association with hernia nuclei pulposi).

Reischauer speaks of "einer auffallend verdickten Spinalwurzel über dem Prolaps, deren histologischen Befund enttauscht".

Bozsik (p. 255) provided a survey of investigations into histological changes of the posterior roots associated with sciatica. He does not report any signs of inflammation, either from the literature or from his own enquiries (33 cases). Bozsik found marrow-sheath degeneration, multiplication of thin fibres, interstitial tissue proliferation, cell infiltrates and hypersensitivity to Sudan stain in the intradural root fibres. Sometimes the changes were found in association with 7 ("oedematous") thickened roots and also with undistended roots, with and without a disc lesion, hyperaemia or neural loss symptoms. As, moreover, the objects of this enquiry were intradural root fibres, we cannot expect it to be very instructive in respect of the condition of the extradural root.

Wijers examined for us fibres removed at rhizotomy from 3 thick and 17 normally thin roots. The rhizotomies were performed *in* the root sheath;





Ectopic ganglion (Pt. L 20456)



Ectopic ganglion cells in nerve root outside the ganglion

hernias were not found. Nothing abnormal was seen upon examination, notably no signs of inflammation (see p. 493).

We were once surprised by the diagnosis "peripheral nerve tissue and ganglion tissue". An *ectopic ganglion* several mm from the dural sac had caused



Tumor tissue invading the root sheath (neurofibroma)



Cystic root

the thickening of this root. On another occasion ectopic ganglion tissue was found in an undistended root.

Three times we found *tumour* tissue invading the root sheaths from the dural sac. The myelographic data and examination of the cerebrospinal fluid had prepared us for this diagnosis pre-operatively and provided the indication for intradural exploration.

Cystic root distension is recognisable at first sight. The root fibres float in the fluid like intradural fibres are seen to do; injected liquid (Novocaine) runs without hindrance into the dural sac; the root can be emptied by squeezing it. (This should not be confused with the perineural root cyst, which presents an entirely different picture. Its site is more peripheral, close to or above the ganglion and almost exclusively in the sacrum.)

The *abnormal course of cauda fibres* was visible during intradural exploration in cases of distended roots.

Two caudal roots run in their intradural course towards the same thickened extradural root We found an intradural root which first plunged into the wide sheath of the L.5 root and then returned to vanish for good in S 1.

Some cauda fibres cannot resist "changing roots", even extradurally.

The finer shades baffle description or cannot be reproduced in photographs Sometimes the fibres had been dissected out before they could be photographed. Root irritation was usually out of the question, because Novocaine had been injected before the variant had been discovered.

We have no ready explanation for the thickening of a root that is not red; it seems to us that in a number of cases it is to be ascribed to a variation in the course of the fibres in the caudal roots. It is evident in the root anomaly due to a common site of emergence for two roots; why, then, should it not be plausible in other cases? A difference in thickness is (in those cases) a different course of the fibres. These anomalies occur perhaps a little more commonly in association with true transitional vertebrae, but considerably more frequently in conjunction with other anomalies of the spinal column, such as pseudo-transitional vertebrae, *spina bifida occulta and a wide vertebral canal. We regard this latter in particular* as a state of transition between the triangular lumbar vertebral canal and the flat, wide sacral shape.



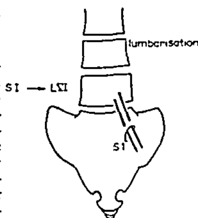
On the analogy of transitional vertebrae, we call the varying cauda fibres *transitional fibres.* This phenomenon might account for the variations

in the neurological innervation patterns. Such of these variations as could be accounted for by transitional fibres are also apparently liable to occur even if there are no clear signs of transition in the spinal column.

[illegible]

effect that muscle and bone tissue is formed at an early embryonic stage, before peripheral nerve tissue can be detected.

What we are concerned with, however, is not *how and when the material of the vertebra is laid down*, but its morphology. The shaping might be induced by nervous elements, however; in that case, if the caudal root in question contains transitional fibres, variations will occur in the neural patterns and morphological anomalies will ensue.

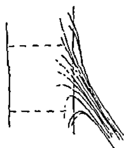


Why, it might be objected, does the root (S.1) belonging to L.VI, in a genuine lumbarisation, *not* impart a sacral shape to this vertebra, if the same root (S.1) – normally emerging at a lower level – is supposed to determine the sacral shape of the S.I vertebra then associated with it? But this vertebra is not innervated by the root running underneath it. Von Luschka – repeatedly checked over by others – described the nervi sinuvertebrales as the nerve tissue which, together with other small branches, supplies the vertebral canal more especially. Now, it appears that these vary considerably; they may drop two or three levels in the vertebral canal before they come into contact with the vertebra they supply. Perhaps these variants of von Luschka's fibres may be taken to be transitional fibres. It might be possible to find out whether the varying descent of the fibres in the vertebral canal corresponds to the type of transitional vertebra in the case in question

After all, a morphogenic function similar to that implied here is ascribed to dysp[illegible]
myel[illegible]
that t[illegible]
cisely what we mean. For, in its "descent" from the spinal cord, does not the caudal root already carry fibres in a definite arrangement? The possibility of the prior existence, in the foramina, of transitional fibres is not affected by the participation or otherwise of the fibres in the moulding of the vertebrae through whose foramina they pass. In a case of that kind, the L.5 root, let us say, would not contain *only* fibres of the L 5 segment in its entire course from the spinal cord; therefore its "composition" must be "determined" in the spinal cord. (But the afferent fibre system grows towards the spinal cord We shall be reverting to this presently; see p. 305.)

Hence the phenomenon of transitional fibres should have its source in the segmental foundation of the root; in fact, more remotely still, *i.e.*, in the

spinal cord. In the presence of anomalies, therefore, we should be justified in speaking of a *micro-myelodysplasia.*



In 1937 Lopes Cardozo showed that in dogs and certain other animals each rootlet also receives fibres from cells not corresponding to the level at which it leaves the spinal cord. "The extensive overlap which has been found for the filamenta radicularia is probably not only of peripheral, but chiefly of central nature" (p. 57). "... the dorsal filamenta ... are more independent in cats than in dogs, which agrees with the smaller overlap found in cats in comparison with dogs".

In his study, Cardozo approached the same phenomenon from an entirely different quarter, *viz.*, from the spinal cord, with the support of anatomical, experimental and physiological research. The agreement between his and our conclusions does seem to lend colour to the assumed existence of so-called transitional fibres in the roots of the cauda equina.

Afferent root fibres are known to spread over more than one segment. Discussing spatial summation of sensory stimuli in man, Droogleever Fortuyn writes (p. 567): "As was shown many years ago by Cajal, incoming fibres of the posterior root spread over more than one segment by means of collaterals. His statements have a special bearing on well-myelinated fibres of large calibre" "Ranson (1913, 1914) traced unmyelinated fibres ... entering the spinal cord .. through the tract of Lissauer into the substantia gelatinosa Rolandi

Szentagothai-Schimert, using the method of 'bouton de dégéneration', was able to follow unmyelinated fibres Each root spread to its own segment and to some segments more proximal". This was also known to Foerster (p. 244): "Der unmittelbare Kontakt der Fasern einer einzelnen hinteren Wurzel ... findet vornehmlich in dem entsprechenden Segmente selbst statt. Doch lassen sich auch *Beziehungen zu Vorderhornzellen höherer und tieferer Segmente* feststellen."

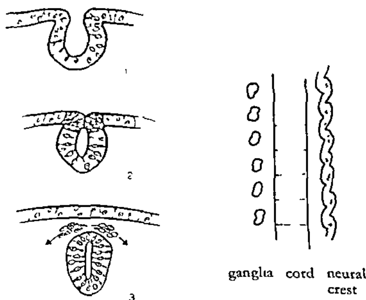
We have been considering the possible existence of sensory transitional fibres only, but the motor fibres show a similar tendency and we shall discuss these later.

We are aware of having over-simplified the phenomenon of transitional fibres by representing it as the result of micromyelodysplasia, since *embryology* teaches us that sensory fibres do not descend, but, after the formation of the spinal ganglia, grow out from the neural crest towards the spinal cord. It may be rewarding to dwell upon the details for a moment. Is there by any chance

some connection between: variations in the morphology of the vertebrae (transitional vertebrae); the arrangement of the neural crest in ganglia; the morphology of the vertebrae and the grouping of the ganglia? If so, can reasons be shown why the arrangement of the vertebrae should be dependent on the induction of the nerve tissue? The answer would have to take account of the developmental process of vertebrae and spinal cord roots and their mutual relationship, *viz.*,

How are roots formed?
How does the segregation of the neural crest take place?
How are vertebrae formed?
How is their relative position determined?
What factors are likely to be involved?

It appears that the views expressed in the recent literature on embryology (Patton, Starck) have changed little from those held by Corning (1925).

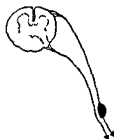


All nerve tissue develops from the ectoderm of the neural plate. While this is being transformed into the neural tube, some of the cells remain between the dorsal part of the neural tube and the superimposed ectoderm. This mass of cells divides into two strands (neural crest) which gradually migrate towards the ventro-lateral aspect of the neural tube.

The genetic connection between the neural crest and neural tube not only explains the connection which develops between elements of the neural crest and the neural tube, but also the relation between developmental disturbances

of neural crest and neural tube. The segmental accumulations of cells which develop into the cerebrospinal ganglia derive from the neural crest. In the fifth to sixth foetal week (Ariens Kappers, p. 791), central offshoots emerge from the ganglion cells, running to the spinal cord (the sensory root), and peripheral offshoots grow towards the end-organs in the relevant segment (the peripheral nerve). Fibres (the motor root) grow from the ventral part of the neural tube along the appropriate ganglion and, once past the ganglion, they form, together with the peripheral offshoots, the peripheral nerve. On their way between the spinal cord and the spinal ganglion, the motor and sensory fibres connect up, segment by segment. We have no exact information as to the order in which these events take place. They occur during the first weeks of embryonic life. The only definite assumption we have the right to make is that the subdivision of the neural crest into ganglia must have taken place before the definitive moulding of the vertebrae, since the spinal ganglia are situated in the intervertebral canals, hence separated from each other by parts of the vertebra. They could not be if the neural crest had not previously been divided up.





The following questions, though by no means fully answered yet, may help us to a better understanding of transitional fibres:

(a) How do outgrowing fibres find their destination?
(b) What are the forces that govern the moulding of the parts of the body?
(c) What do we mean by segmentation?

A rough outline of the views expressed in the literature will have to suffice.

After their migration, the neural crests at first lie very close to the primitive spinal cord. Owing to the fact that the growth of the other derivatives of the germ-plates is quicker than that of the central nervous system, the body (skeleton, muscles, organs) descends in relation to the spinal cord, this movement often being erroneously called the ascent of the spinal cord. The roots grow together with the descensus (being only hypothetically outstretched). Along their common route, the motor and sensory bundles remain together.

Remembering that the primitive spinal cord and the "accessory" spinal cord are contiguous at first, it is easy to see how a fibre growing centripetally reaches the spinal cord. There are some who suppose that this fibre growth begins simultaneously with the segregation of the neural crest, or assume that straggling cells show the way – still clearly outlined, so



recently after the separation, between the cells of the surrounding tissue. According to Held and Harrison (see Corning), the neural crest furnishes the material for a syncitium into which the axis grows, as though into a pre-formed path.

What these proponents hold in common is that a fibre growing towards the centres is, essentially, merely restoring the communication between two already interrelated neurogenic tissues. Seen in this way, the sensory root is the link between two tissues which have possibly passed through the same developmental modification.

Definite evidence as to how a peripheral nerve makes contact with its end-organ is not to hand. In the current view of experimental neuro-embryologists, this process not only involves an intrinsic pattern of growth of the nerve tissue, but also the development of the periphery, of the skin and underlying structures (Weisz, 1952, Starck, p. 334). Gegenbaur (see Corning) thinks that the nerve fibre grows together with the end-organ from the outset. In experimental animals, His and Hensen (see Corning) demonstrated the growth of peripheral nerve elements which could not yet have been in contact with their end-organs. Long strips of protoplasm were thought to serve for pre-formed tracts, while the induction of, say, an extremity on the nerve tissue growing towards it was effected by mechanical or chemotactical stimuli. This view seems to rule out any influence of the nervous system upon the moulding of the end-organs. Harrisson, on the other hand, proved the induction of the central upon the growth of the peripheral nervous system. The impression one receives is that both the central nervous system and the end-organ induce the growth of the peripheral nervous system. This is corroborated by recent studies of experimental animals by Miner (1957), who also found that another operative factor was the period of the animal's development.

Little is known with certainty about the influence of the nervous system upon the moulding of parts of the body. Nevertheless, the reciprocal inductive action of end-organ and central nervous system shows that not only the morphogenesis of the parts, but also the normal development and function of the peripheral nerves depend upon the central nervous system (Corning, p. 532). In other words, a micro-myelodysplasia (by which we here mean a slight modification in the structure of the myelum productive of transitional fibres) might affect the build-up and formation of the peripheral nerve tissue and the end-organs supplied by it.

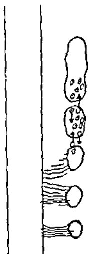
Segmentation

Bolk (p. 4) says: "A segment is the primary morphological component of the bodies of vertebrates".

Detwiler (1934, p. 439) says: "There is no evidence in support of the view

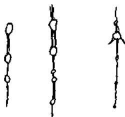
that nervous metamerism is intrinsic, at least in the spinal cords of the forms which we have studied".

Segmentation is, largely, an artificial division of the body into parts, yet, this artificiality notwithstanding, it is incontrovertible. The spinal cord (and the brain) seem to be unsegmented; nevertheless, indications of segmental structure are to be seen both in them and in the extremities.



Several rudimentary segments are already visible in a human embryo less than 2 mm in length. These somites multiply proximally and distally, with the result that an embryo about two months old has approximately the same segmentation as that seen in an adult. Together with other factors, the notochord – itself unsegmented – is held to be responsible for this segmentation. It is beyond doubt that the first rudimentary segments are present before the first ganglion-like agglomerations appear in the neural crest; the supposition that segmentation is induced by an antecedent division of the primitive central nervous system is untenable. We are therefore confronted with the fact that the neural crests are broken up into something like segments; or, to put it differently: the cells agglomerating to ganglia on the borders of such an intervening space have to "choose" between one or other ganglion for their habitat.

Looking at drawings of embryos at this stage, it will be seen that the sensory roots run in an unbroken line from these ganglia to the spinal cord. The transitional fibres phenomenon is potentially inherent in the arrangement of the cells from the neural crests into ganglia; also in the outgrowth of the closely contiguous sensory fibres. That this potential inherence is by no means imaginary may be illustrated by an analogous case. The build-up of the sympathetic nervous system is likewise thought to proceed segmentally. Not only are the cervical ganglia found to have conglomerated to a few knots of nerves, but practical experience shows that the anatomy of the prevertebral ganglia and of the truncus sympathicus is wholly chaotic. Seldom does one find an orderly segmental build-up, variation proves to be the rule over and over again. Strict sympathetic parietal metamerism is lacking (Van Gelderen, p. 331)



It is a known fact that the boundaries of a vertebral segment do not coincide with those of a vertebral body, the latter being composed of half a sclerotome



at a higher level and half one from a lower level. This being so, the spinal ganglion is seen to lie in the *centre* of a segment, an observation that may help us to visualize how the cells of the neural crest are distributed among the ganglia. Through cell division, material for new segments is formed at the proximal and distal ends of the neural tubes; Starck (p. 364) finds an almost unlimited production in some animals. Theoretically, therefore, material is superabundantly available for the formation of excessively large ganglia, say in the lumbo-sacral region where much neurogenic tissue is required for the extremities. The organism, however, prefers an exact apportionment of tissue compatible with the function required of it, *i.e.*, an opportune proportion. On these grounds, segmentation during ontogeny might be defined as an "opportunist" process of distribution that has proved useful (or the optimum) in phylogenetic development.

In some molluscs and insects the whole organism can be controlled from a few nerve plexi. In vertebrates, however, a given function is assigned to the spinal ganglion forming the centre of some particular segment, for which a certain number of cells are allocated to that ganglion. The inference from this opportune allocation would seem to be that the distribution of the neural crests in ganglia is a fairly random one, secondary to (quantitative) factors in the remainder of the segment. And let it be said that neither the ontogeny, nor the function of the nerve tissue in a given segment suggests that an arrangement of the neural crests in "ganglia" precedes segmentation or induces it. It is, on the contrary, far more probable that the initially unsegmented neural crests come to be so arranged as the result of the general induction to segmentation; possibly, too, the distribution of the cells among the ganglia is variable, not to say haphazard.

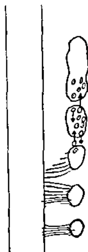
Quite unexpectedly, we found considerable support from the following publication for our hypothesis, which is that a discrepant arrangement of neural crests in larger or smaller ganglia might be responsible for irregularities in the distribution of the sensory fibres among the (caudal) roots, hence also for malformations of the body. Byl (pp. 99 and 121) described a dysrhaphic embryo of 5½ months. In addition to several other deformities, chiefly in the lower half of the body, he reported a spina bifida occulta "extending from L.I caudad. The spinal cord was strongly grown together with the dura, starting from L.I; towards caudally the *segmental structure could not be distinguished*



Sketch, illustrating Byl's description

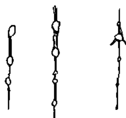
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numerical correspondence between the ganglia and the cartilaginous neural arches, regardless of the number of somites" (1936, p. 164).

In a word Detwiler's view is that the somites induce the segmentation of the neural crest (which was Bolk's point of departure (p. 8) as far back as 1910). The bulk of the inducing extremity is a determinant for the number of spinal nerves (in relation to this extremity) and an atypical distribution of those spinal nerves (transitional fibres!), if any, makes no difference to the over-all functions of that extremity. (Weiss, Hamburger, *ibid.*, see Starck, p. 364.)

Once the neural crest is segmented into spinal ganglia, the distribution thus obtained is important to the morphogenesis of the vertebra (arch). This points to the relationship between (anomalous) transitional fibre and transitional vertebra.

So far, we have only come across lumbo-sacral transitional fibres. It would not surprise us if they occurred only, or most frequently, at the level of the (cervical and) lumbo-sacral intumescence. The increased rate of cell division in the composition of the cervical and lumbar somites might well lead to "mistakes" in this cell division. To illustrate what we mean, we would recall that there are seven cervical vertebrae and an eighth cervical root; there are five lumbar vertebrae, the fifth often being difficult to distinguish from the first sacral as a transitional vertebra. The dermatome overlap does not take place across the axial lines, in the arm, the C.4, C.5, C.6 and C.7 dermatomes are sharply separated by the axial line from the Th.1, Th.2 and Th.3 dermatomes, or in the leg: L.2, L.3, L.4 from S.1, S.2. The position of C.8 and L.5 in relation to the axial lines is uncertain (Biemond, p. 17). Personally, we do not believe that this coincidence is accidental. In popular parlance, these roots are "all at sea". Even physiologically they are in a state of transition; hence at these levels there is a predisposition to anomalous transitional phenomena.

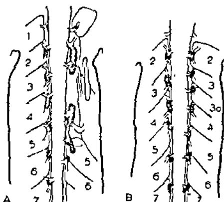
Transitional fibres may vary the sensory innervation patterns to greater or less extent. It has been shown that a certain root can be seen clinically to perform the whole function of its neighbour; and we have also reported cases in which it did so only partly. The obvious reasoning is to relate the intensity of these variations to the number of fibres grouped in an unusual manner as the result of this neural transitional tendency.

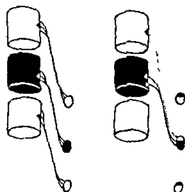
Lastly, we come across numerous minor variants in the clinic which are undoubtedly within the physiological range. The literature offers no satisfactory explanation for these, in current opinion they are due to an extension or development of corresponding nerve elements varying from individual to individual. Contrary to this opinion, we suggest that even these minor variants

well. . . . In a mass of nervous tissue several *big spinal ganglia could be recognized*".

We believe that transitional fibres are offshoots from those cells which migrated to a neighbouring ganglion when the neural crests were dividing up.

We found even greater support in Detwiler's papers (1934, pp. 395 and 1936) It would be





A, graphic reconstruction of spinal cord, ganglia, nerve-roots and myotomes in an Amblystoma embryo, showing incomplete segmentation of ganglia on the right side following excision of somites 2, 3, 4 and 5. B, similar reconstruction of an embryo in which the three right brachial somites (3, 4, 5) were replaced by a graft containing four somites (7, 8, 9, 10) from another embryo. Note presence of a supernumerary ganglion and nerve (3A) associated with the additional myotome. Redrawn from Detwiler, *Neuroembryology* Copyright 1936 by the Macmillan Company and used with their permission.

superfluous to quote him *in extenso*, but we would mention that in Amblystoma he demonstrated experimentally the relation between the somites, the segregation of the neural crest into ganglia, the number of spinal nerves and both the moulding and number of vertebral arches. Among other things, numerically related disturbances were brought about in the above tissues and organs by the extirpation or addition of somites. Here are some of his conclusions: "The maximum extent of the limb rudiment serves as an index of the spinal nerves which shall contribute to the plexus" (1936, p. 166). "Segmentation of spinal ganglia and associated nerves is subservient to mesodermal metamerism. There is no evidence in support of the view that nervous metamerism is intrinsic The number of (brachial) nerves can be increased experimentally . . . Limbs typically supplied by three nerves may exhibit normal function when supplied by one nerve or by five" (1934, p. 438). "Abnormal segmentation of the ganglia . . . may interfere with the subsequent morphogenesis of the neural arches" (1934, p. 433). "There is typically a

finer nerve rami in the skin, we find it very hard to believe that a systematic crossing of the roots would have escaped his notice while carefully dissecting the plexus.

On the other hand, it would be inconsistent to doubt the existence of transitional fibres simply because Bolk found none, since their ramification is thought to be situated in small fibres of the same dimensions as those held in the literature, on Foerster's authority, to be anatomically undissectable.

Definite information on the course of nerve fibres through the plexus can be obtained by analytical dissection. It is instructive to recall the opinion of the investigator who did this in the human body. Bolk (1910, p. 24) does not consider plexus formation to proceed from the intrinsic development of the peripheral nervous system, but to be a phenomenon brought about by mechanical causes. In other words, the shifting and displacement of end-organs govern, mechanically, the composition of the plexus. Detwiler's experiments (transplantation of extremities with retention of function, in spite of the fact that the spinal nerves passed atypically through the plexus) provide physiological corroboration of the opinion Bolk arrived at on anatomical grounds.

Having shown that there is a causal relationship between the condition of a given root and a phenomenon in an end-organ, a fibrous connection between the two may be assumed, the manner in which this fibre runs through the plexus being immaterial. Put differently: Provided the phenomenon in question occurs *via* an intact fibre connection, or through the cutting of that connection, physiologists considering the communication between root and end-organ may legitimately disregard the plexus. And indeed, this happens repeatedly in practice.

For general comments in the following discussion of the *dermatomes*, we refer to Foerster's statements concerning this matter (see Bumke, Part. V, p. 246).

	Dermatome determination		*Overlapping*
Bolk	In man	Anatomically	Little or none
Head	In man	Experiences from pathology	Little
Sherrington	In apes	Physiologically	Marked
Foerster	In man	Remaining sensibility after nerve root section above and below	

are due to an individually different fibre distribution; this will roughly follow the same pattern, on the understanding that a root is likely always to contain a fibre or two belonging to its neighbour. By rough standards, this last condition produces the innervation patterns with which we are familiar and which we call physiological. Accordingly, it is right to distinguish between "physiological" and "anomalous" transitional fibres, the latter producing variations striking enough to be called anomalies.

What would the function of the *plexus* be in this matter? Some authors, *e.g.*, Armstrong, ascribe the occurrence of anomalies to marked variations in the plexus. We know of no indications which would substantiate this view. Bolk's studies seem rather to point to the contrary. This anatomist succeeded in tracing the fibres from the root to the associated dermatome right through the plexus. Sherrington and Foerster also ignored the plexus in their physiological experiments.

This apparent tangle of fibres need not have been an obstacle to the physiologist because a theory, resting on known anatomical facts, does not represent the plexus as a kind of switchboard. In the comparison with telephone communications, the lines run in cables from the switchboard (spinal cord) to the receiving instruments (skin, muscles). Instead of running straight on over houses and fields, telephone lines are bundled in cables, from which, without interruption, lines are taken or added to; but they continue to be direct connections. This analogy holds good for those parts of the body where no plexus is formed, *e.g.*, in the thoracic region. The fibres of the peripheral nerve entering a plexus on this busy highway seek their "travelling companion" to proceed together along a well sign-posted road (or detour) towards their destination. But they continue to be the uninterrupted, direct, segmental line of communication between the spinal cord and dermatome-myotome, just as they would if they were to criss-cross as fibres through the tissues from the intervertebral canal to the end-organs. The plexus is the main road along which the fibres travel to their appropriate dermatome or myotome, as the case may be. It is surprising that several authors should try to account for the overlapping of the dermatomes, as established by Foerster and Sherrington, for example, by a systematic crossing of the fibres in the plexus. There is nothing in physiology to suggest that this is so, though it is admittedly a convenient way of graphically representing the overlapping of the dermatomes. This supposed crossing of root fibres in the plexus (Hansen and Van Staa, p. 98) conflicts with Bolk's anatomical observations. If we may accept Foerster's suggestion that it was impossible for Bolk to dissect out the



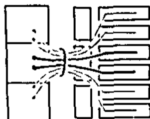
fibres resolve this contradiction? As we have already hinted and as Bolk explicitly stated, we may disregard the plexus and we shall do so while developing the following argument.

We recall that:

Bolk dissected the nervous connection between root a and region x, thus determining the dermatome A^1 belonging to root a and the segment of the spinal cord A.

Foerster cut roots a^1 and c^1; region x in A^1 proved to remain connected to the spinal cord *via* root b^1. These findings are not contradictory; fibres may run from x towards roots a or b though the course of these minute ramifications may not be anatomically controllable. Foerster, like Sherrington, concludes that there is overlapping of A^1 through fibres coming from B^1 and calls the area of maximum fibre extension from B^1 thus obtained the "B^1 dermatome" (belonging to root b^1 and segment B).

Lopez Cardozo (and Rabl; see motor transitional fibres) studied the implantation of the filamenta radicularia in the spinal cord. Roots a, b and c are composed mainly of fibres derived from the opposite segment, but fibres from neighbouring segments join them either extramedullarily or intramedullarily.



We pointed out before that mechanical monoradicular irritation of a root (*e.g.* b^1) produces pain in the innervation pattern projected behind it (dermatome B^1). Upon violent irritation, the pain spreads beyond B^1 (to A^1 or C^1).

Combining these findings with the evidence of extra- and intramedullary connection of the dorsal filamenta radicularia with higher and lower parts of the spinal cord, we speak of *transitional fibres* when the assumption of a fibre connection of the A^1 dermatome *via* root b (alien to the segment) with the part A of the spinal cord (proper to the segment) accounts for the observed phenomenon of pain radiating to an adjacent dermatome.

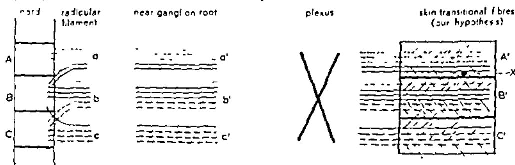
Conclusions

If transitional fibres from A^1 to A *via* b be accepted, it is a plausible argument that region A^1 belongs entirely to segment A.

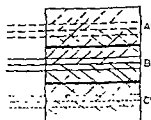
Foerster and Sherrington observed the extension of irritability into adjacent skin areas and concluded from that fact that the dermatomes overlap. The "dermatomes" found by Foerster, however, were not dermatomes at all, but root areas. The boundaries he marked out are the result of taking a root (*e.g.*, b) as demarcating a segment and regarding the innervated skin area

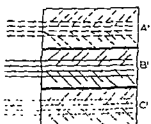
Dusser de Barenne	*In laboratory* animals	*Physiologically*	*Very marked*
Klessens De Boer		Stimulation after application of strychnine	Not applicable to man (?)
Edinger Keegan	In man	Physiologically Hypalgesia from proved single nerve-root loss	Very little or none
	In man	Experiences after rhizotomies	? Unreliable
	In man	Monoradicular mechanical stimulation during operation	? Exact, yet vague limitation

The information elicited varies with the methods employed. By exciting the root during the operation, fairly reliable clues can be obtained as to which vaguely defined innervation area the pain stimulus reaches; but it is our



experience that a clean-cut dermatome boundary cannot be marked out in this way. Each chart thus obtained differs from the other. The disturbances of sensation after the rhizotomy of one root are often too trifling for a close dermatome determination. The method of "the remaining sensibility" depends precisely on leaving one root intact in the middle of an area denervated by extensive rhizotomies This method, which, as applied to human beings, has received some notice, particularly through Foerster's work, produced markedly overlapping dermatomes. They vary from *Bolk's*, the boundaries of which are sometimes rectilinear, without overlapping. Foerster puts this down to the impossibility of tracing the finer nerve rami in the skin





Would the existence of physiological transitional

dermatomes are smaller and more clearly marked off than they showed them to be – the real dermatome limits, after this correction, come nearer to Bolk's and these, in turn, appear to be more consistent with practical findings (Head-Edinger-Keegan, etc.) than Foerster's. On account of his own experience in practice, B. Brouwer (see Foerster, p. 254) was another who did not agree with Foerster's dermatome boundaries; Brouwer believed that Bolk's were more correct.

After studying monoradicular lesions, Keegan found clear-cut "dermatomes" with little overlapping, the dermatomes of L.4 and L.5 not overlapping at all (Lewin, p. 755). It need hardly be added that, in our view, actually these "dermatomes" were the projections of root areas upon the skin. In the vast majority of cases, the lesion of a root (b or b^1) puts most of the fibres belonging to segment B and dermatome B^1 out of action. The exclusion of a comparatively small number of physiological transitional fibres in the injured root will not produce salient neural loss symptoms in the bordering regions with A^1 and C^1, particularly as their innervation *via* transitional fibres in roots a and c has remained intact. That is why Keegan finds smaller root areas than Foerster.

The size of a root area appears to vary with the degree of mechanical stimulation applied to a root during operation. *E.g.*, if root b is mildly irritated, enough for the sensations to be just experienced, these are indicated as occurring in B^1; with more vigorous irritation, its effect upon the comparatively few physiological transitional fibres is experienced by projection of the induced sensations in A^1 and C^1, for which reason the root areas appear to be larger. During a sensory rhizotomy, say in root sheath b^1, involving the examination of minute bundles of fibres one by one, a small group of fibres in which physiological transitional fibres from, say, A, predominate, may possibly be stimulated by chance, the result being radiation of pain projected into A^1. Should these transitional fibres predominate to such an extent that they can legitimately be called anomalous, then, even upon mild irritation of b or b^1 (the whole root, or in a bundle), the radiation of the sensations into A^1 will appear as a variation or anomaly.

The findings of Dusser de Barenne, Klessens and De Boer likewise become comprehensible in the light of the transitional fibre hypothesis. The application of strychnine to the dorsal root would aggravate the irritability of the comparatively small number of transitional fibres quite considerably, right to the smallest ramifications in the boundaries; hence the excessive size of the "dermatomes" found by this method.

It should not be inferred from all this that we wish to make the boundaries of a dermatome or root area subservient to the nature of the method of

behind it as a segmental division of the skin, *i.e.*, dermatome. The reason why Foerster's "dermatomes" are so large is that transitional fibres from segments A and C also run in root b. Foerster and Sherrington have varying "dermatomes" (often overlapping very erratically), but the source of these variations is a haphazard distribution of nerve fibres through the roots. We are with Lopez Cardozo and Rabl in denying that the root (a, b or c) is laid down purely segmentally and, therefore, also that it offers a means of segmental dermatome determination, because the anatomical and physiological evidence argues against it. (*Loc. cit.*, p. 403) (see Brandt: Embryologie, p. 496.)

Foerster, beyond doubt, was aware of the existence of segmental variations in the neural innervation patterns (p 248 *cit.* Sherrington: "die einzelne Dermatome... konnen oralwärts oder caudalwärts verschoben sein"; Hansen and von Staa, p 242, *cit.* Foerster: "individuelle Varianten der Segmentversorgung"). He did not interpret this knowledge when explaining the overlapping of his "dermatomes".
It would seem as though Foerster, although aware of the importance of variations and, perhaps, of the existence of "transitional fibres" in our sense, was yet prepared to accept the theory of varying overlapping to account for them.

The question as to how a fibre from A reaches A¹ *via* an alien root b is in the same category as that other problem, *viz.*, how does the peripheral nerve reach its end-organ? The latter phenomenon is an accepted fact, but there is no explanation for it. It seems to us more likely that a fibre communication is effected through the agency of a transitional fibre from A *via* b through B¹ with its *own* end-organ in the segmental dermatome A¹, than that fibres from B *via* b and B¹ join up with the alien end-organ, which is *not segmentally* allied, in the A¹ dermatome. (Foerster-Sherrington.)

Although, in our opinion, the "classical" dermatomes as described by Foerster are not dermatomes in the true sense, but innervation patterns of *roots projected upon the skin*, they have proved to be clinically valuable for that very reason The clinician wishing to locate a *root* disorder does not seek to know the boundaries of an ailing dermatome; what interests him are the manifestations, if any, of the disorder in the known innervation pattern of that diseased root. However, we have been considering dermatomes, not because of their clinical value, but because the overlapping of the dermatomes or root areas can be plausibly explained by the phenomenon of transitional fibres; our plea for the existence of these transitional fibres gains support from this clinical aspect. Their striking compatibility with the facts about roots and dermatomes we learn from physiology is worth mentioning.

Now that Foerster and Sherrington's intensive overlapping appears to apply to root areas and not to dermatomes – with the result that the true

another peripheral nerve. Does this continued growth take place along the prepared path of anastomoses? Anatomists (including Spaltenholz) do, admittedly, say that skin nerves anastomose; but these anastomoses are comparatively remote from the terminal branches to which Foerster refers. We have no information on intersegmental anastomoses of peripheral skin nerve branches on the border of the dermatomes; it is inferable from the sketches in textbooks that the authors are doubtful as to the existence of these anastomoses. "The terminal ramifications of every main fibre . . . were found to remain independent from those of other fibres" (Weddell, p. 170, see Granit, p. 43). "There is no contact between fibres of two segmental nerves" (Bolk).





(b) Even if tractless growth were admitted to the terminal ramifications of peripheral nerves, it would not be compatible with the current theories on the induction of peripheral nerve growth to suppose that they would cross the frontier into a dermatome foreign to them.

(c) Very little degeneration is observed in the part of the spinal cord situated opposite a cut root. Yet the ascending degeneration, central to an interrupted neurite, is an established fact. If all the fibres of a cut root belonged to one "segment" of the spinal cord, signs of extensive degeneration should appear in this "segment" before the denervated root area could be re-innervated by fibre growth from neighbouring root areas.

These difficulties can be resolved by interpreting the facts within the terms of the transitional fibre hypothesis·

(α) It is unnecessary to invoke the tractless growth of nerve endings, since fibres from segment B were already present in "dermatome" B¹ (*via* roots a and c) before the section of root b

(β) If the function of these reserves (transitional fibres *via* roots a and c to "dermatome" B¹) were too weak, they could "learn" to function more powerfully. If they need to grow to cover the whole of the denervated dermatome B¹, that growth takes place in the dermatome proper to those fibres, which accords with the views on the induction of the growth of the peripheral nervous system.

(γ) Although the ascending degeneration in the central nervous system remains quantitatively the same, it will not be confined to that part of the spinal cord which is situated opposite to the cut root. Owing to the transitional

investigation employed. It does become clear, however, in the light of our postulate, that smaller root areas are found by methods based on the root (Edinger, Keegan, Head, root stimulation, neural loss after rhizotomies) than when the *largest* area is sought in which skin stimuli are observed (Foerster, Sherrington, De Barenne), for, by the latter procedure, the fields of projection of the transitional fibres in the adjacent dermatomes are also scanned, or even deliberately sought.

Is an exact determination of dermatomes feasible at all? Not, we should say, by the methods we have been discussing. If the "method of the remaining sensibility" is complemented by section of the extramedullary filamenta radicularia to the adjacent spinal cord segments, this leaves the intramedullary transitional fibres (Lopez Cardozo), which means that only somewhat greater exactitude is achieved

We might further point out that our subject is almost entirely confined to affections of the *retroganglıar sensory fibres* in the root sheath between the ganglion and dural sac. Section of these fibres causes irreversible loss of function of the fibre involved, as retroganglıar regeneration has not been seen to occur A root area thus denervated can, therefore, only be re-innervated *via* other fibres (*e.g.*, in other roots); there is a marked difference between this and the re-innervation of a sensory denervated region behind a *peripheral nerve lesion*; we shall not dwell on the continued growth of the fibre which is possible in this case. We shall deal with the re-innervation or regeneration of *the motor fibres* in the root sheath after a *lesion* on page 399.

How, in this light, are we to regard the *re-innervation* of a root area desensitised by retroganglıar rhizotomy? The current opinion is that the terminals of the nerve tissue grow out of the adjacent "dermatomes" into the denervated "dermatome". "Sensory axons can produce collateral sprouts following degeneration of neighbouring nerve fibres" (Edds, see Woolf, p. 1506). By physiological standards there are no objections to this view. Through-growth of peripheral terminals has been seen microscopically, even in implanted skin. Yet this conception contains certain inconsistent elements, which we shall try to demonstrate by the following arguments.

(a) A process of growth, *un*verified in the anatomically dissectable peripheral nervous system, is in that case assumed to take place in the extremely minute, anatomically undissectable peripheral terminals. The branches of the peripheral nervous system invoked to explain this re-innervation are so attenuated that, according to Foerster, even Bolk was unable to trace their course. It is not sound reasoning to assign an anatomically unverifiable property to the terminal branches which the remainder of the peripheral nervous system is known not to possess. For, transplantation of a nerve only produces satisfactory results after an end-to-end anastomosis on the preformed paths of

For further illustration we submit the following examples.

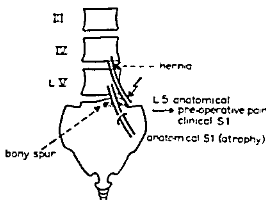
Patient ♀ B. 5756. A year previously a *total rhizotomy* of the right S.1 root had been performed elsewhere. When the patient was admitted to our clinic, we were unable to detect any neural loss symptoms. Upon exploration it was visibly evident that the S.1 root *had in fact been severed completely*. After total section of the left S.1 root, there was no evidence, either immediately or later, of motor or of sensory disturbances!

We are forced to assume that a sufficient quantity of S.1 fibres from L.5 and S.2 were present in both S.1 root areas to deputise fully for S.1.

The following is from our records, without further particulars:

Immediately after section of one of the S.1 roots, marked sensory loss symptoms were presented (which never cleared up entirely) and an over-all paresis of the calf muscles, which diminished after two months. In this patient there must have been very little intersegmental fibre overlapping in the S.1 root.

Normal spine (7 C + 12 Th + 5 L). *Patient H. 14257. 4 years previously*: Pain r. leg. Where? Reflexes? Sensory disturbances?



Ach. refl. reduced to negative.

Myelography: Recess L.IV – L.V.

Operation: Fresh hernia L.IV – L.V.

Upon monoradicular irritation of the anatomical L.5 root, pain felt as pre-operatively in lateral edge of foot and little toe (clinically S.1).

Note To the right of L.V – S.I there was a margin of bone, evidence of a healed disc lesion or hernia nuclei pulposi; the disc still protruded slightly on the left; root S.1 on the right was atrophic and unresponsive to stimulation.

Conclusion. Lesion of the S.1 root caused by old hernia L.V – S.I on the right. Assumption of function of the sensory S.1 fibres by the S.1 fibres in L.5?

Notes from first observations of an unknown patient (further details lacking).

Normal spine

7 years previously Clinical history recorded complaint of pain in lateral edge of foot and little toe.

Sensory disturbances: Lateral edge of foot and little toe Reflexes?

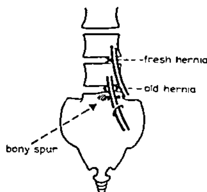
Symptom-free

Now. Pain in hip, big toe.

Sensibility intact!

K r Reduced to negative. A.r. +!

Operation: Old ossified hernia L.V – S.I. Fresh hernia L IV – L.V.



fibres the signs of degeneration will be spread over a wider area; moreover, the most affected part of the spinal cord itself receives transitional fibres from neighbouring roots. Hence the degeneration will be more diffuse.

These considerations suggest that the peripheral area behind a root lesion is re-innervated by fibres already belonging to the affected part of the spinal cord and root area and that the phenomenon of the transitional fibres which we have brought forward plays an important part in this. Transitional fibres also possibly come into action (temporarily) during re-innervation after a peripheral nerve lesion, but that is an entirely different problem and we shall not deal with it here.

How is re-innervation after a root lesion reflected in the clinical facts? Sometimes pre-operative sensory and reflex disturbances disappear within two weeks after the surgical intervention. This must be due to the recovery of the *function of fibres not anatomically injured*. In some of the cases, no residual neural loss symptoms were found at check-ups two months later. If residual symptoms did not clear up till six to nine months after the intervention, we assumed that they had been the result of an *anatomical lesion* of the root fibres.

After selective sensory rhizotomies we have 1) very occasionally had extensive sensory disturbances, but 2) generally they were slight, and 3) by no means seldom they were never mentioned or revealed by the patient nor observed objectively. However, when loss of sensation was brought about as above, 4) the symptoms never again cleared up altogether, to our knowledge; but, after six to nine months, 5) the denervated areas had become smaller, or the insensibility changed to hypalgesia.

The views expounded above on re-innervation by transitional fibres offer a reasonable explanation for this clinical experience.

We may sum this up briefly as follows Re 1) few or no transitional fibres, re 2) physiological, re 3) many or verymany transitional fibres Re 4) occurrence of sensory disturbances after retroganglıar selective sensory rhizotomy proves that the neighbouring roots have no reserves (few or no transitional fibres) to take over the function of the severed fibres at once This accounts for the persistence (for months) of sensory disturbances occurring after a sensory rhizotomy. It is vain to expect regeneration of the severed root fibres themselves, because this is not seen to occur after *retroganglıar* section Therefore, if the sensory disturbances abate six to nine months later, it must be due to intervention *via* neighbouring roots. Re 5) the few transitional fibres in adjacent roots can *learn* to perform the function of the severed fibres, either by functioning more intensively, or by invading the fallow (denervated) skin area proper to the segment.

Permanent sensory disturbances provide evidence of the complete lack of transitional fibres.

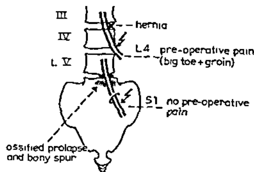
areas without asking ourselves of what fibres the root is composed. Anomalous transitional fibres, however, may produce such marked variations that it is impossible to establish a localising diagnosis with certainty upon the evidence of neural symptoms. An interpretation of possible variations may explain the incongruity between several clinical facts and the diagnosis will become less doubtful. In this climate, clinicians will be less tempted to label symptoms which do not comfortably fit into the rest of the neurological picture as "psychogenic". From the fact that variations may be brought about by anomalous transitional fibres it follows that local anaesthesia is desirable. It is from the reaction of the patient to irritation of a root that some degree of certainty can be gained as to whether the abnormality found in that root is likewise responsible for the disorders to which surgery is being applied.

E.g., Myelography L.V – S.I pos., L.IV – L.V neg.; obvious hernia at L.V – S.I. Irritation of root L.5 during operation is felt as the pre-operative pain.
Exploration at L.IV – L.V also reveals a small bud-shaped hernia under and in root L.5.

Examples now follow to show how the interpretation of variations could have prevented, or did prevent, a "recurrence".

Pre-operative pain in posterior aspect of thigh and hollow of the knee (S.2).
Myelography: L.IV – L.V pos., L.V – S.I neg.
Operation: L.IV – L.V hernia.
Irritation of L.5 produced pain in little toe, lateral edge of foot (S.1).
L.V – S.I not explored.
Recurrence Same pain as before.
Re-operation: Hernia at L.V – S.I.
Discussion. The discovery of the variation (L.5 = clinical S.1) should have been considered as a warning that the vaguely described pain at the back of the thigh (S.2) might possibly have something to do with a lesion of S.1, *i.e.*, a hernia at L.V – S.I.

Normal spine Patient B. 5257.
Pain in big toe and on the left side of *groin*
Sensory disturbance in lateral aspect of calf.
A r. and K r. reduced
Motility left leg reduced all over.
Radiograph. L.IV – L.V narrowed.
L III – L.IV narrowed.
Spondylosis L.III – L IV.
Myelography L V – S I neg.,
L IV – L V neg.,
L.III – L.IV neg. (or ±).



L.5 painful upon pressure (radiation unknown).
S.1 thin, atrophic; *not painful upon pressure.*
Conclusion. Earlier acute syndrome of S.1 "cured".
Re-innervation of the S.1 root area *via* the still atrophic S.1 is unlikely.
Assumption of the S.1 function by the S.1 fibres in L.5 is unlikely, because the fresh hernia under L.5 did not induce an S.1 syndrome.

Was the S.1 function *taken over by S.1 fibres in S.2?*

Why do sensory disturbances result from a temporary *minor lesion of uninterrupted root fibres* (through a hernia, say,), whereas they sometimes do not occur at all and sometimes only to a slight extent after total section of these fibres? Why do existing sensory disturbances sometimes *diminish* after section of the injured fibres involved? How are we to account for the lack of sensory disturbances in the innervation area of a root in spite of its having become so thin and atrophic (as verified at operation) that it would seem to be incapable of functioning any more? The point in common in these clinical experiences is that, the more radically the direct communication between periphery and spinal cord is cut, the easier it seems to be for "reserve" fibres (running in other roots) to take over the functions in a denervated root area. We found no answer to these questions in the literature. Maybe comparison with the following facts will point the way to a solution.

After extirpation of the thoraco-lumbar sympathetic trunk (Th.12 – L.4), a sympathetic denervation of hip and leg sets in. In a matter of days to weeks – within which period nerve tissue cannot reasonably be expected to regenerate, nor, in fact, has it been shown to do so – large tracts of this area proved to have been sympathetically re-innervated. This re-innervation was prevented by section of the motor roots Th.12 and L.1. Ray and Console attributed this re-innervation to sympathetic reserve fibres in these motor fibres and called them residual pathways of the sympathetic system. They were said not to come into operation until the direct main lines of communication had been interrupted for some time, this in view of the temporary denervation after the section. It seems to us quite reasonable to assume that similar residual pathways also exist in the peripheral nervous system; only, seeing that transitional fibres take up a *function*, we should then prefer to speak of a "residual function" (loc. cit. p. 328).

The phenomenon of physiological and anomalous transitional fibres provides a useful theoretical background, with the help of which the physiology of the root can be better understood.

In practice, physiological transitional fibres will not be of much account, because our attention is directed to possible neural loss symptoms in root

areas without asking ourselves of what fibres the root is composed. Anomalous transitional fibres, however, may produce such marked variations that it is impossible to establish a localising diagnosis with certainty upon the evidence of neural symptoms. An interpretation of possible variations may explain the incongruity between several clinical facts and the diagnosis will become less doubtful. In this climate, clinicians will be less tempted to label symptoms which do not comfortably fit into the rest of the neurological picture as "psychogenic". From the fact that variations may be brought about by anomalous transitional fibres it follows that local anaesthesia is desirable. It is from the reaction of the patient to irritation of a root that some degree of certainty can be gained as to whether the abnormality found in that root is likewise responsible for the disorders to which surgery is being applied.

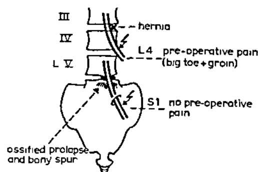
E.g., Myelography L.V – S.I pos., L.IV – L.V neg.; obvious hernia at L.V – S.I.
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Examples now follow to show how the interpretation of variations could have prevented, or did prevent, a "recurrence".

Pre-operative pain in posterior aspect of thigh and hollow of the knee (S.2).
Myelography: L.IV – L.V pos., L.V – S I neg.
Operation: L.IV – L V hernia.
Irritation of L.5 produced pain in little toe, lateral edge of foot (S.1).
L.V – S.I not explored.
Recurrence: Same pain as before.
Re-operation: Hernia at L.V – S.I.

Discussion. The discovery of the variation (L.5 = clinical S.1) should have been considered as a warning that the vaguely described pain at the back of the thigh (S.2) might possibly have something to do with a lesion of S.1, *i.e.*, a hernia at L.V – S.I.

Normal spine. Patient B. 5257.
Pain in big toe and on the left side of *groin*
Sensory disturbance in lateral aspect of calf.
A.r. and K r. reduced.
Motility left leg reduced all over.
Radiograph. L IV – L.V narrowed.
L III – L.IV narrowed.
Spondylosis L.III– L IV.
Myelography L V – S.I neg.,
L IV – L V neg.,
L.III – L.IV neg. (or ±).



Diagnosis: Hernia L.IV – L.V (?)
Operation: Removal of L.5 arch.
Exploration: L.IV – L.V: Nothing unusual.
L.V – S.I: Old ossified prolapse.

Monoradicular irritation of the root above this bony prominence (S.1, clinically S.1, lateral edge of foot and little toe).

From this it follows that the S.1 root does not conduct the pre-operative pain The roots exhibit *no* tendency towards variation.

For these reasons the L.V – S.I disc is left intact and the exploration is extended to L.III – L.IV: hernia; typical indication of pain upon irritation of L.4.

Discussion When re-examined, the patient was without symptoms. Unnecessary weakening of the spine by clearing L.V – S.I was avoided. The extension of the exploration to a third disc was motivated by the definite absence of root variations.

Patient 3 Slight low back pain.
Pain radiating to hip and lateral edge of foot.
Myelography. Dubious L IV – L.V.
Operation Slight prominence L IV – L.V. Not extirpated.
No changes in disc at L.V – S.I.
Irritation of anatomical S.1, reaction in hip and lateral edge of foot.
Sensory rhizotomy S.1.
Post-operatively and at check-up: Still slight low back pain.
No radicular pain.

One should always be on the look-out for variations in the innervation patterns if there are neurological or myelographical indications of a L III – L.IV syndrome. This seems to have a greater propensity to occur in association with transitional vertebrae, when variations and transitional fibres are anything but rare.

A correct interpretation of the transitional phenomena will not infrequently lead the practitioner, after further consideration, to name the level where the abnormality was found, not L IV – L V, but L III – L IV. The L.III – L IV syndrome is usually poor in symptoms, for example, the absence of a positive Lasègue and of classical neural loss symptoms does not necessarily provide evidence against the existence of a hernia at that level

Provisional summary on sensory disturbances

A pattern of sensibility is an empirically established correlation between the sensibility in an area (of the skin) and one root; it is used in the clinical location of root lesions by comparing the extension of sensory disturbances with existing charts Many of these charts differ one from the other; minor, but also considerable individual variations are not rare. For this reason, the facts obtained by sensory examination lose some of their value for the location of a root lesion. Accordingly, there are authors who attach little importance to the results of their own examinations in this field.

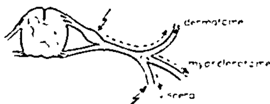
Indications of many variations in the course of the fibres of the cauda

equina were obtained from the interpretation of our clinical data; they can be accounted for by the phenomenon of transitional fibres, to which we ascribed a "residual function" within the context of re-innervation.

Depth sensation – Referred pain – Head's zones

To summarise briefly, we shall cite some data from the literature which have become common knowledge.

The irritation of a posterior root produces sensations of pain projected to the skin. (Foerster, p. 291, Sherrington, Head and many others.)

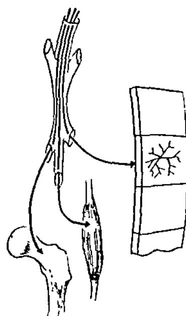


Irritation of the skin by the "remaining sensibility" method produces evidence that the sensory innervation of the skin is partitioned off into root areas, called dermatomes by Foerster and Sherrington.

Kellgren has demonstrated by subcutaneous injections that the muscles, bone and ligaments are also innervated in a metameric distribution. (Armstrong, pp. 10, 82, 84, Bradford and Spurling, p. 108, Lewin, p. 635.)

In agreement with Gaskel, Hansen and von Staa report indications of a metameric segmental innervation of the viscera.

There is dissociation between the nerve supply to the skin and that to the subcutaneous organs (Foerster, p. 303, Kellgren quoted by Bradford, p. 108), by which is meant that the skin and the organs lying immediately under it need not necessarily be innervated by nerve tissue from the same segment. "In any event, a given myotome does not necessarily occupy a position immediately internal to the *corresponding* dermatome" (*Kellgren* and Lewis, see Sinclair, Weddell and Feindel, p. 198).



The recognition of segmental innervation of the myotome enabled Armstrong (p. 84) to draw up a chart, with the aid of which he utilised disturbances in the innervation of a myotome for locating a root lesion. Admittedly, he adds that the areas thus marked off overlap and are not exact.

Schmorl (p. 172) gives the names of many authors who are persuaded that a root lesion at the same time induces disturbances of autonomous nervous elements.

Sensations initiated by stimuli applied to visceral fibres can be projected into the remainder of a root area and may be expressed by hypersensitivity in the associated dermatome. It is on this, in part, that Head's dermatome determination is based (Foerster, p. 249).

Kellgren (Bradford and Spurling, p. 108) noted the projection of pain stimuli from a myotome-sclerotome to the associated dermatome.

Depth sensibility (loc. cit. p. 282) is the ability of subcutaneous tissues to experience sensory stimuli. They are experienced tardily in a vaguely defined area, which the patient locates with the palm of the hand.

By contrast, irritation of the superficial sensation of the skin produces a sharp reaction, which the patient has no difficulty in locating clearly with his finger. Depth sensibility is usually associated with the muscles, ligaments and joints. In the general sense, the ability of the viscera to react to stimuli is in the same category.

The receptive power of the subcutaneous tissues is also said to be limited to the perception of certain qualities of sensation, Head's classical grouping being roughly followed, thus.

Superficial sensibility: (protopathic)	Sense of pain. Sense of temperature. Fine tactile sense (tickle)
Depth sensibility. (gnostic)	Sense of postural movement. Sense of localisation, writing numbers. Sense of discrimination. Sense of vibration, reaction to faradic current. Gross touch, pressure.

This classification is not commonly used in modern physiology on account of its incorrect nomenclature and incompatibility with the more refined methods of examination

A myotome often proves to be supplied from a root innervation other than the dermatome lying immediately above it, a good example being the sensory innervation of the superficial and deeper parts of the inguinal zone. The superficial sensibility is supplied largely by roots L.1 and L.2, whereas the deeper parts of the groin, notably the muscles and ligaments, are supplied chiefly by roots L.3 and L.4 respectively (Spaltenholz).

	Nerve	Root
Superficial sensibility "groin"	Nervus ilio hypogatricus	L.1
	Nervus ilio inguinalis	L.1
	Nervus genito femoralis	L.2
	Nervus lumbo inguinalis	L.2
	Nervus cutaneus femoralis lateralis (L.1) L.2 (L.3)	
Depth sensibility "groin"	Nervus femoralis [L.1] (L.2) L.3 L.4	
	Nervus obturatorius [L.2] (L.3) L.4	

Possibly efferent and afferent fibres reach and leave the same myotome along common pathways, but little appears to be known about the manner in which these fibres are connected to the spinal cord. Foerster (p. 309) repudiates Lehmann's view that the afferent fibres supplying depth sensibility run *via* the motor anterior roots only. Although, in his opinion, it is an established fact that some of the depth sensibility as well as the sensory innervation of the visceral organs are supplied by the anterior roots, there is no justification for reversing that fact and assuming that all these fibres run *via* the anterior roots. Foerster persists in his opinion that the majority of the afferent fibres, also for depth sensibility, reach the spinal cord *via* the posterior roots.

According to Bell and Magendi's law, all afferent fibres pass through the posterior roots. Magendi himself was the first to doubt this. Foerster (p.305) summarises the arguments in favour of an afferent pathway *via* the anterior root as follows:

1. Faradic stimulation of the central part of a severed anterior root is felt as pain in the associated peripheral innervation area.
2. Re-innervation can occur after section of a posterior root. Permanent sensory denervation ensues only if an anterior root is severed as well.
3. The presence of afferent fibres in the anterior horn was also demonstrable by histological analysis. After section, an ascending degeneration is found in the spinal cord, while both Foerster and his collaborators found ganglion cells in the anterior root belonging to an afferent fibre. This turned out not to be exceptional and was found most commonly in the anterior roots of S.1 or S.2.

Foerster thinks that many of the afferent fibres in the anterior roots are probably sympathetic fibres. He associates these with re-innervation after section of the posterior roots. He had also noticed the variability of the sensibility exhibited upon re-innervation. In the majority of cases, the sensibility returning after section of a posterior root bears the character of depth sensibility. It continues to be distinguishable from normal depth sensibility (*i.e.*,

the receptive power of subcutaneous tissues) by a higher stimulation threshold, a longer latent period, the indeterminate localisation and diffuse distribution of the sensations experienced. Accordingly, it is natural to regard the returning sensation as depth sensibility and to attribute it to the coming into action of the afferent pathways in the anterior roots.

Foerster was unable to find any sensory disturbance after severing numerous anterior roots, in contrast to the initially complete anaesthesia and analgesia after severing several posterior roots. This experience led Foerster (p. 308) to propound a "*main pathway*" for afferent fibres (*via* the posterior roots), both for depth sensibility and superficial sensibility, and a "*side track*" (*via* the anterior roots) for the afferent fibres of both kinds of sensibility. He suggested that the side track would not come into action until the main pathway had been cut. This idea fits in roughly with the residual pathways in the sympathetic system postulated by Ray and Console.

Thus, as far as the afferent fibres are concerned, we have the following systems:

1. *Afferent main pathway via posterior roots.* After section of one or two posterior roots, the first reserve system comes into action.
2. Intensified functioning of the *transitional fibres in the still intact neighbouring roots.* We call this phenomenon the *residual function* of the transitional fibres in the neighbouring roots.

In his experiments, Foerster severed several posterior roots lying side by side, *e.g.*, from thoracic 12 to sacral 2 inclusive. Hence it was impossible for transitional fibres in these areas to perform their residual function. After the complete section of the afferent main pathways, the side tracks have an opportunity to come into action.

3. *Afferent side tracks in the anterior roots* according to Foerster (identical with the residual pathways in the sympathetic system).
4. After comprehensive denervation (section of posterior roots, anterior roots and local sympathetic system), signs of re-innervation occur very sporadically, for which only the activity of *pathways via sympathetic plexus* at a distance – *e.g.*, along the blood vessels – could be responsible (Foerster, p. 307). This view is refuted by Sturup and Arnold Carmichael (p. 216). They show that in their experiments in man the peri-arterial plexus did not serve as an afferent pathway for pain from the periphery. Similar views are expressed in the literature (Busch, Coates, Davis and Pollock, Woollard).

The precariousness and variability of re-innervation are due to this multiplicity of possibilities. One pathway only begins to unfold when the preceding main pathway has been more or less cut off.

Foerster gives several examples of the varying circumstances under which sensory re-innervation occurs. Among other things he states (p. 308) that re-innervation takes place sooner and more completely in those cases of posterior root section in which the intervention was undertaken to relieve long-existent (years) severe pain. This accords with our own findings, *viz.*, that, after sensory rhizotomy (for the relief of pain) of a root in whose area sensory loss symptoms were presented pre-operatively, sensory re-innervation took place sooner and more comprehensively than after a similar treatment of a root in the area of which there had been no pre-operative sensory disturbances. Foerster tries to account for this by assuming that the central nervous system had already been thrown into an extreme state of irritation before the section by the permanently incoming afferent stimuli. We are more inclined to think that several afferent pathways were failing in their function, due to the previously existing lesions, so that, after total section of the posterior root, the side tracks were already prepared to take over the function of the main pathways.

By "*referred sensibility*" (see p. 358) we intend to convey the awareness of sensations in a different part of a root area from that to which the stimulus is applied. Thus, for instance, the irritation of a posterior root will project the sensation of pain into the skin, but also, though less acutely, into the muscles, or even into the viscera. As far back as 1910 Bolk (p. 11) spoke of the intersegmental radiation of pain and predicted that this phenomenon might well lead to interesting observations in the clinic once we had fuller knowledge of the segmental innervation of the internal organs.

Some authors, the Anglo-Saxons more especially, make a distinction between referred superficial sensibility and referred deep sensibility. (Literature: White and Sweet.) As against this, the expression "*referred pain*" is quite often used for the awareness of pain somewhere in a root area produced by a stimulus *elsewhere* in the *peripheral* part of the same root area. More specifically the term stands for sensations of pain projected upon the skin by stimuli applied to visceral organs. Through ignorance of the facts, this has led to the attribution of low back pain in an unreasonably large number of cases to disorders of the abdominal organs. The incorrect interpretation of the data in the publications of Mackenzie, Head and Knotz have added to this confusion in no small measure (see Hansen and von Staa). This does not take away from the fact that their publications furnished valuable material for a better understanding of the physiology of the root and that their observations deserve to be better known than they actually are. In 1894 Head drew up the first well-founded plan of human "dermatomes". This was based on the following observations:

1 Head regarded the area of eruption of a spinal ganglion affected by herpes

zoster projected upon the skin as the image of the segmental innervation of the skin. He called the areas thus observed "dermatomes" and this would seem to be incorrect; for, these areas of eruption represent the results of trophic disturbance. In the current state of our knowledge it is premature to regard these areas of eruption as the complete projection of all the fibres running through the spinal ganglion. Moreover, in that case, it would be a matter, not of a dermatome, but of a spinal ganglion projection, or a root area. It is, therefore, not surprising to find that Head's "dermatomes" are smaller than those of Foerster and Sherrington, who determined the utmost extension of the total root area. Nevertheless, the circumscribed, smaller "dermatomes" of Head come closer to modern conceptions than the larger "dermatomes" suggested by Foerster.

2. For dermatome determination Head used the hyperaesthetic zones which occur after disorders of visceral organs. He was aware that "referred hyperaesthesia", or hypersensitivity, occurs in a corresponding segmental area of the skin. Skin stimuli which are normally not painful then cause pain in such an area.

3. *Head's dermatome determination depended further upon the data obtained* from transverse lesions.

The occurrence of a dermal hypersensitivity in the corresponding segment is a remarkable fact which has been observed repeatedly. We do not know whether it helped Head much to determine the dermatome or root area, but we are rather inclined to doubt it; because, more remarkable than the phenomenon itself is the fact that these hypersensitive zones are so often assumed to occur in the L.4, L.5 and S.1 root areas, whereas not a single author mentions the supply of visceral organs by these roots (Bohm, Franksson and Petersen, p. 48; Delmas; Goldthwait, Lewin, pp. 635, 870, 874; Foerster, pp. 290, 286; Dinko Sučić, p. 56; Van Gelderen, pp. 329, 331; Spaltenholtz). Head is no exception in this, apart from taking the prostate as being supplied by the S.1 root, whereas other authors suggest that the vegetative fibres for the prostate run *via* S.2 or S.3. It therefore seems improbable that hypersensitivity zones should occur in the L.4, L.5 and S.1 root areas as the result of stimulation of the viscera. For the same reasons, low back pain in the projection fields of the latter roots is unlikely to be referred pain originating in diseases of abdominal organs But we should not lose sight of the fact that such communications do exist between roots S.2 to S.5 and the genito-urinary tract.

Bohm *et al.* (p. 48) point out in a monograph that pain is projected into the small pelvis as the result of lesions to these sacral roots and state that radicolysis or rhizotomy does sometimes clear up this pain. They even ask themselves

whether some morbidity of these sacral roots could not produce secondary changes in the organs of the small pelvis. (?)

Opinions vary in the literature as to whether low back pain can be due to referred pain from the abdomen. In addition to Bohm's statement as above, we quote what Lewin says (p. 870): "Diseases of the genito-urinary tract have a definite bearing on low back pain. The genito-urinary system is frequently overlooked as a possible factor in the production of back pains". On page 874 the same writer protests against gynaecological operations for the relief of low back pain, saying: "The gynecologist must be very careful in recommending pelvic operation for low back pain, because in many cases the pain persists after operation". Goldthwait is likewise of opinion that gynaecological and urological diseases are seldom the cause of low back pain and of radicular pain.

Hypersensitivity is probably the most familiar expression of referred pain. There are other forms of hyper-irritability of the (peripheral) nervous system as the result of a stimulus applied elsewhere, from among which we take the following:

Hyper reflex action

Increased muscle tone (Mackenzie, Hansen and von Staa). This is accounted responsible for the spastic rigidity of the long back muscles.

Hypersensitiveness to cold. According to Zukschwerdt, cold and damp do not initiate neuritis. Owing to an existing lesion, neuritis or neuralgia, a particular innervation area is highly irritable and this makes the individual hypersensitive to cold at that place.

Hyperhydrosis, segmental secretion of sweat, pilo-erection and vascular reactions are known (Head, Mackenzie). We have seen several examples of it in sufferers from low back pain, a very clear one being that of a young girl with a degenerative back, *i.e.*, wide spinal canal and spina bifida. At the level of vertebrae L.IV and L.V there was a hypertrichotic area the size of a hand's palm, where the skin was permanently wet.

Vasomotor disturbances in the legs. Epstein and Lewin saw pseudodysbasia intermittens associated with epiduritus spinalis. Verbiest shows more satisfactorily a connection with vascular disturbances from the epidural space in association with stenosis vertebrae. Reischauer (p. 8) suspects a disturbance of *para*sympathetic fibres in the fifth lumbar root. He saw this vascular disturbance only when there was a lesion in this root. The argument against this view is that the *para*sympathetic fibres are not thought to run above the level of S.2, nor, as far as we are aware, have they been shown to. (Sympathetic fibres do

not enter into the question at all if, on the authority of competent authors, we accept the fact that L.2 is the most caudal lumbar root through which sympathetic fibres run.)

A vertebral stenosis almost invariably occurs at L.III and L.IV; thus the associated vascular disturbances can scarcely be accounted for by a lesion of root L.5. According to Reischauer, the vascular disturbances persist after a lesion of L.5 has healed. It is precisely after an exploration in the epidural space that they occur and usually they clear up in anything from six to nine months, while neither a motor nor a sensory disturbance need necessarily have occurred during the whole of that period. In that time the vascular tissue and accompanying nerve tissue regenerate in the epidural space. This is apparent from the fact that low back pain, which ceases after the rigorous clearance of vessels in the epidural space, is apt to return after this period.

Furthermore, these vascular disturbances also appear to occur in association with monoradicular lesions of the S.1 root; it was then verified that a lumbosacral hernia was not simultaneously interfering with the L.5 root. The most plausible explanation of these vascular disturbances in the legs still is a reflectory disturbance in the collateral circulatory system according to Leriche, as the result of irritation of epidural vascular nerve tissue.

Superficial hypersensitivity (and other excessive irritability) may be revealed in various ways, *viz.*,

(a) Locally, in "segmental" distribution (Head), *i.e.*, probably in a single root area.

(b) Contralaterally. This is rare, but when it occurs it does so in the corresponding root area

(c) In a neighbouring root area. We know of cases of monoradicular S.1 lesions verified at operation, with sensory disturbances in the S.1 root area and diminished Achilles reflex; at the same time the knee reflex had intensified or hypersensitivity existed in the S.2 root area. Superficial hypersensitivity in the projection field beside an injured root is by no means rare. Maybe it is responsible in part for the inaccurate and variable indication of sensory disturbances and radiation of pain as the result of a monoradicular lesion.

(d) In the homolateral part of the body. The distribution of sensory disturbances across a large area is usually ascribed to an adventitious neuritic factor or is sometimes understood as pointing to the psychogenic aetiology of the complaints. This is soon suspected of patients who, after suffering from low back pain, begin to complain of pain in the homolateral arm. This doubt would understandably arise in the case of a patient who, for example, indicated the same sensory disturbances on the right-hand side in the supine position as

were experienced on the left-hand side in the prone position. But on the whole it remains to be seen whether there be not an organic basis for the distribution of sensory disturbances through the homolateral half of the body and the possibility should be taken into account in practice.

The following considerations may be apt here:

(1) Occasionally radiating pain is indicated following an entirely unknown anatomical pattern (*e.g.* from the diaphragm or deep in the abdomen, a short, sharp stab of pain along the groin, the genitals, the inside aspect of the thigh down into one of the toes). They are different from lancinating pains, while there are no signs in the otherwise healthy person of radiculo-neuritis and the reactions to lues in blood and cerebrospinal fluid are negative. Comparable disorders are discussed and explained by Weizsäcker (p. 10). He points out the encroachment of an organically caused sensory or motor functional disturbance upon adjacent innervation areas not organically disturbed.

(2) In other persons a curious correlation is to be found between the patient's personality structure and heightened sensitivity after surgical treatment of the nervous system, while the changes brought about in the nervous system can in no way account for the origin of those sensations on any known anatomical basis.

(3) There is also a correlation between the cessation of pain in one part of the body and the occurrence of identical pain in another part. Syndrome shift provides a ready explanation for this in psychosomatics. On the other hand, in certain cases one and the same disease (*e.g.*, cervical and lumbar osteochondrosis) will be responsible for the varying localisation of syndromes; but we are referring to those cases in which such diseases do not occur generally.

"Summation" of stimuli in the synapses of the spinal cord and in the thalamus, "sensibilisation" by the sympathetic nervous system, "generalisation" through changes in the central nervous system under the influence of chronic local stimuli, etc. (Hansen and von Staa, Gagel, von Frey, Langly, Foerster, Head, Mackenzie, etc.) are among the suggestions put forward to account for these mysterious syndromes. They are phenomena, the existence of which cannot perhaps be denied, but as explanations they could no longer be vindicated at the bar of modern physiology (Laruelle, Wendel).

Let a few comments suffice, since further digressions would take us ever farther from our subject.

The spino-thalamic pathway of von Monakow discovered in animals has not been demonstrated as an anatomical system in man. The 1200 fibres which can be counted in man are not enough to serve the complicated function formerly ascribed to the "pain path" in antiquated postulates. There is sup-

posed to be an interplay between various pathways in intramedullary-intracerebral synapses. The view that there are different receptors for different qualities of sensation is gradually being abandoned. The forms we know of are alleged to be modulations of one and the same organ. The demonstrable existence of thick and thin nerve fibres no longer implies that one conducts protopathic and the other epicritical qualities of feeling. It has been established beyond doubt that thick fibres conduct stimuli more quickly than do thin ones. This, among other things, led to the hypothesis that the various qualities of sensation arise from a difference in the conduction of stimuli; thus sensations of pain would be the result of disharmonious conduction of stimuli. This modern view is opposed to that held by Foerster, who assigned the various qualities of sensation to individual fibre structures; thinking along these lines, he supposed that certain fibre structures were hypersensitive to certain noxious elements. The modern view is that sensations of pain can only be induced in areas supplied by fibres which carry stimuli at different speeds. Up to the present we have not been able to detect under the microscope any arrangement of thick and thin fibres in the root right in front of the ganglion. All we know is that, on stimulating sensory root fibres (macroscopically visible) just in front of the spinal ganglion, one very occasionally succeeds in finding small sensory bundles that are not painful; on the whole, however, all the fibres of a sensory root are painful under pressure, provided the stimulus be sufficiently intense. "...Any specific stimulus if sufficiently intense will cause pain" (Von Frey, see Granit, p. 44).

Ranson (see Foerster, p. 242), on the other hand, has shown an arrangement of the fibres according to thickness in the root close to the spinal cord; thickly myelinated fibres are said to run in the middle and to conduct tactile stimuli, while thin fibres are arranged laterally and conduct pain stimuli.

Ranson's observations have been checked up by modern physiologists. The arrangement in a certain order is regarded as a preliminary alignment of the fibres, before they ascend in the pathways of the spinal cord, thus: The thick fibres assemble before entering the posterior tracts, whereas the thinner ones group together in order to turn at once towards the lateral tracts. The location of these systems, which are so important to vital, vegetatively autonomous, epicritical functions, is the same in the older and more recent literature. Nowadays, the function, *e.g* as regards the awareness of pain, ascribed to them is not that of conducting pain, the action of these fibres and systems is considered to be conditional upon disharmonies in the conduction of stimuli of such magnitude that they produce the sensation of pain. After section of that part of the spinal cord in which we think the ascending pathways of these

systems are, the awareness of pain is said to cease because, after successful severance, only those pathways remain which conduct one and the same kind of stimulus. Disharmonies will, however, continue if the section has been less felicitous or if the nerve-ends of severed pathways are over-stimulated. The spontaneous causalgic or parapathic sensations liable to occur after chordotomy are said to result from this.

We know of three patients in whom the disease followed the same course. After a sensory rhizotomy, numb patches arose on the skin and the pre-operative pain disappeared. At a later date chordotomy had to be resorted to for the relief of pain in the contralateral leg After this, all three patients were free from pain for ten to fourteen days, after which causalgic pain arose, spontaneously and by touch, in the patches that had been numb and painless prior to the chordotomy. All three patients led a neurotic existence, accompanied by conversions in one case. This did not surprise us, as we assume a neurotic structure of character to be a condition for causalgia. For all that, we consider the burning pain to be organic in origin, caused by a change in *function* of the central nervous tissue, since not all the fibres severed by the chordotomy are of one kind or thickness. Verhaart *et al.* (p.159) proved this, finding an *undifferentiated* degeneration of ascending thick and thin fibres in the spinal cord after chordotomy. According to the above theories, discordances could thus arise and pain be induced.

As we see it, discordances in the conduction of stimuli were, in the three cases mentioned above, the result of disturbed function in the chordotomised spinal cord and were responsible for the causalgic pain felt in the previously painless, numb patches.

If anything emerges from this summary, it is that we still know little with certainty about the course of the fibres and physiology of the pathway systems, the suggested explanations proffered, in particular, still being unsatisfactory. We have a new hypothetical "explanation" to add to the list, in spite of the pitying smile of the experienced physiologist we shall thereby be provoking. It is not that we overestimate the value of this hypothesis – which will probably suffer the same fate as that of so many others – but, by submitting it, we shall at the same time be drawing attention to an often neglected phenomenon. Moreover, it will afterwards become clear why we considered it necessary to bring forward certain facts from the realm of physiology when discussing sensory disturbances.

This hypothesis can be illustrated by the following two cases taken from our practice, examples which could be multiplied many times.

Patient S. ♀ *12457*

Slightly b-

symptoms These complaints cease spontaneously at the end of another two months.

Patient W. ♂. 21257.

Nothing abnormal in structure of character. Mild spondylosis of the spine. Typical complaints and symptoms of hernia L.V - S.I on the right. No complaints or symptoms in back or legs after extirpation Six weeks later: continuous nagging, severe pain in right arm and neck, without neural symptoms. The complaints vanished spontaneously at the end of another two months.

Both patients had taken bed rest for a few weeks prior to the operation, during which time these new complaints did not occur; they could not reasonably be ascribed to the patients' immobilisation (7 and 4 days respectively) after the operation.

What had actually happened was that intense chronic pain stimuli had abruptly ceased; subsequently mysterious sensations of pain were experienced in a homolateral extremity, without a known anatomical substrate and unaccompanied by known neurological syndromes.

We have seen that the functional re-innervation – not due to nerve regeneration – proceeded differently in areas subjected chronically to intense pain stimuli (Foerster and our own observations). We have already adduced evidence from the literature and our own observations of the existence of afferent subsidiary pathways, which come into operation when they are needed, *i.e.*, in proportion as the inhibiting influence of afferent main pathways declines. Whatever we may call the process (summation, generalisation, sensibilisation, residual function), *changes take place in the nervous system causing it to function differently*; to our mind it is immaterial where they occur, whether demonstrable organic changes are involved or whether only the function changes.

What happens when the initial cause of these changes is (abruptly) removed? A great deal depends upon the answer to that question. Suppose that the main pathways of the afferent system were so seriously injured that secondary paths came into operation. When the lesion has been removed, the main pathways begin to function again, so that main and secondary pathways are functioning simultaneously; yet by definition and as an experienced practical fact, main pathways put a brake on the activity of the secondary pathways. Presumably the result will be discordance between the pathway systems.

Modern physiology states that disharmony in the conduction of stimuli lies at the foundation of sensations of pain, hypersensitivity, aggravated irritability and so forth. It is not by mere chance that pain begins to be felt in the homolateral extremity when the period of quiescence has elapsed, after which a decompressed root usually begins to function again, as can be verified by the recovery of motor and sensory functions.

The phenomenon described above could be explained by the simultaneous

disharmonious functioning of two pathways, causing hyper-irritability.

Put in very simple terms, we might imagine that the spinal cord, embarrassed by the superfluity of stimuli, side-tracks them to other areas. The obvious area to "send them to" is that built up in the same way (intumescentia cervicalis or lumbalis) as the area which is the source of the surplus of stimuli. It could also be supposed that over-irritability will make itself felt in an area where a number of abnormal stimuli were already arriving on account of still subclinical morbid changes and would have remained below the pain-stimulus threshold if there had not been that additional irritability.

It is also a theoretical question whether the severe pre-operative pain in one extremity had perhaps masked a less intense pain of longer existence in the other extremity.

For our purposes it is immaterial which of these postulates is correct. The main thing is that an organic origin is *possible!*

The above considerations may serve as a warning against over-hasty decisions as to the psychogenesis of hypersensitivity and of the extension of zones of pain to the trunk, the extremities and the head.

SYMPTOMATOLOGY - NEUROLOGICAL SIGNS

Further Reflections on Sensory Disturbances

In the first part of this Chapter we have been considering the sensory disturbances consequent upon a known lesion of the nervous system which can be detected objectively in a corresponding known innervation area. The occurrence of variations from this interrelationship has already been discussed in detail and we have suggested more than once that these constitutionally determined variations or anomalies should be regarded as manifestations of a total state of being, which we call the degenerative back.

These objectively verifiable disturbances due to morphological variations are *distinct from disturbances in the perception of stimuli. Phenomena*, moreover, occur which are unmistakably conditioned morphologically, yet the relationship between their morphology and function cannot be proved convincingly.

As some insight into these disturbances of perception and mysterious phenomena will help to clarify the description of the degenerative back as a state of being, we shall consider them in the following pages with reference to the views expressed in publications by Buytendijk, Granit, Laruelle, Prick, Stenvers, Walshe, Weddell, White and Sweet.

Reflections on Sensations of Pain and Neurological Signs and Symptoms

I. *Psychology*

A. Inadequate sensations, false reference of stimuli, false reference of sensations

The awareness of sensory experiences is a conscious, non-volitional act of the psyche. A strong will can, by dint of practice, acquire the power to moderate or vivify the experience. Body and soul constitute a totality, the two-in-one character of which is eminently exemplified by the functioning of the central nervous system. It is a logical statement that nothing can be experienced without an active central nervous system.

An idea of the mental process entailed in the perception of stimuli can scarcely be conjured up without at least a modicum of philosophic reasoning. The concepts *soul*, *principle of life* (= *life*) and *psyche* are either classed under distinctive definitions or are identified with each other, all according to the school of thought to which the user of the word is committed. We shall not concern ourselves with these fine distinctions.

Seeing that the body is the integrating morphological and physiological pattern of our spiritual life, we consider the soul (principle of life, psyche) to be the sum total of our behaviours, *i.e.*, the complex of experiences of all our bodily activities. To put this differently: The psyche is the experiencing aspect of our bodily phenomena, *i.e.*, our attitude towards life, which is our behaviour. Meaning it in this sense, we shall from now on only use the word "psyche".

A *perception* is the awareness of a physical activity. *How does an undisturbed perception come into being*, regardless of where and when? If we take the perception of pain, for example, and here in particular, we could say that it is an unpleasurable experience. Unpleasurable sensations and their counterpart, pleasurable sensations, are the basic sensations from which all other, more differentiated, feelings derive. There are different aspects of every feeling, including that of pain, which may be said to be a psychosomatic total reaction to the impingement of pain-provoking stimuli from the environment. Owing to the activity of pain receptors, afferent (pain)nerves, posterior horns and spinothalamic tracts and their extension in the brain stem, also to the activity of the thalami optici, thalamo-cortical communications and certain areas of the cerebral cortex, we ultimately become aware of the pain-producing situation as a differentiated unpleasurable sensation.

If we analyse these sensations of pain psychologically, we find that there is a certain intent (orientation), answering to the formula "away from", associated with a specifically appurtenant and adequate vegetative reaction pattern (secretion of sweat, pupil reaction, palpitations and so forth), acting as the incarnation of the intent just referred to.

It is not enough only to analyse the feeling of pain; an endeavour should be made to grapple with and analyse the painful event. Inherent in the latter is the cognitive and conative experience of the pain-producing situation, or, expressed differently, knowledge of the source of pain and the affective reactions to it. Hence the emotional event encompasses the affectively charged knowledge of the relevant pain-producing situation in the "world about us".

It follows from all this that, under normal circumstances, knowledge of the external situation and the appurtenant affect with which that knowledge is charged are closely integrated. And it is precisely the affective charge of

knowing which contributes so much to the retention by the known of its objective reality value. For example, say that one presses a seal on the skin of a blindfolded person. The description this person can give of the seal (large, *uneven, warm) is in effect simply the affectively charged knowledge of the* body indented by the seal. The body refers the pressure, *i.e.*, the infraction, with all attendant circumstances, to which it is subjected. Similarly, seeing a tree is, essentially, placing that tree (through certain media) into the eye, *i.e.*, an impression (of light) in the retina. This means that the body, through its structure and activities (morphological and physiological properties) always acts as the reference system for the psychic behaviour; in this case this being "experiencing".

Prerequisites to the awareness of a sensation are said to be: a stimulus, conduction of the stimulus and an intact central nervous system in a conscious individual for the experience to be possible at all. Apparent exceptions to this simple rule appear, upon closer scrutiny, to be illusory.

(a) Perception without a stimulus
Silence is heard; the absence of light stimuli produces the sensation of black. The subsidence of stimuli or the alteration of continuously acting stimuli acts as a stimulus.
(b) Perception without transmission of stimuli.
The absence of a part of the body and the numbness behind a severed nerve are experienced, even without touch, through lack of the tonic-afferent stimuli formerly issuing therefrom Consciously missing is experiencing that what was before no longer is. In cases of extensive lesion of the cauda equina, the patient may feel as though the lower extremities did not belong to the body; upon recovery, however, the sensation of the legs' again "belonging" is often experienced dramatically. Both an afferent link and its lack act as a stimulus and are experienced.
(c) Experiencing sensations without awareness is a contradiction in terms. Unconscious and subconscious (as well as hyperconscious) perceptions are absolute examples of the imperfect assimilation of stimuli.

Inadequate, i.e., unequal, disparate, ill-balanced.

If the quality of a perception is incompatible with the quality of the stimulus, the perception is said to be inadequate.

We recapitulate the prerequisites for the actuation of a perception:

Primarily a stimulus coming from the outside world

The reaction of the receptor to the stimulus.

Intact transmission both to and in the central nervous system.

Normal activity of the central nervous system enlisted for the assimilation of the stimulus.

It is necessary to bear in mind that any one of the above components may be disturbed, with the remainder functioning normally.

If the receptor "strikes a wrong note" (*e.g.*, on the reinnervation of a sensory nerve), or the transmission of the stimulus modifies the impulse out of all recognition (*e.g.*, causalgia), we speak of *false reference of stimuli*. In that case the perception of the stimulus presented to the brain need not itself be disturbed, but appears to be inadequate to the stimulus. Hence the perception may be adequate to the incoming stimulus ("Erregung") but inadequate to the initial stimulus.

In the strict sence, *perception is inadequate* when the impulses presented to the brain, after somatically perfect assimilation of the stimuli, are interpreted incorrectly. Given an adequately experienced sensation, we still have to allow for the possibility of its being imparted incorrectly or awkwardly to the environment; this is called *false reference of sensations*.

We now have to ask: What factors are likely to be conducive to the inadequate perception thus formulated? Arranging these in a certain order, we get the following chart:

1. Disturbance of the receptors and their transmission of the stimulus to the sensorium.
2. Disturbance in transmission, *i.e.*,
peripheral nervous system,
central transmitting pathways,
subcortical areas for the assimilation of the stimulus,
transmission between subcortical and cortical areas,
cortical activities,
integrative activity of the cortico-sensory projection fields and the emotional brain,
emotional brain (limbic system), *e.g.*, after leucotomy,
associative coupling of the cognitive aspects,
affective charge of the cognitive content
3. Disturbance of the perception,
resulting from abnormal psycho-functional conditions (psychogenically inadequate experiences)
4 Distorted representation to the outside world.

Re 1. Changes in the receptive field. Even under normal conditions, sensations of pain do not depend upon a specific pain stimulus. Every stimulus which causes pain to be experienced *is* a pain stimulus, it is not the stimulus but the experience which is inadequate if a sensation of pain results from what is usually a specific tactile stimulus. "Any specific stimulus, if sufficiently intense, will cause pain" (Van Fray, see Granit, p. 44, Glees, p. 77). Put differently: Every stimulus which, if only remotely, hints at the possibility of damage to the tissue, can become a pain stimulus Heat is experienced as tepid, warm or hot, but "very hot" alerts the modulation of feeling felt as pain. A pain signal

is not sent out for every noxa; *e.g.*, there is none for very cold. If the cold is pathogenic, pain is ultimately nevertheless felt owing to tissue damage. The signals sent for tissue damage or for danger are sometimes called "nociceptive" and belong to the protopathic system; they evoke defensive and evasive reflexes, are transmitted *via* thin, unmyelinated fibres, or of low myelin content, and have a *high* stimulus threshold (Ariens Kappers, p. 807). It was assumed that a separate "nociceptive receptor and transmitting system" existed for these most primary protopathic stimuli, but Granit denies it.

Under certain (pathological) conditions, specific stimuli of non-pathogenic intensity for other modalities of feeling may "serve" as a pain stimulus and produce sensations of pain. *E.g.*, heat or deep pressure can be experienced as pain in a partly reinnervated or regenerated area of an injured root or peripheral nerve.

A receptor need not specifically register stimuli of a certain sensory quality; pressure on the eye produces sensations of light, even in the dark; some sounds can be painful; transversal sound vibrations elicit shudders. Accordingly, in modern physiological parlance there are various modulations of one and the same sensory receptor which are capable of converting various stimuli under different conditions into currents of action varying in intensity, duration and velocity. This view only seems to conflict with Woollard's statement (p. 367), *viz.*, "In general it may be put forward that pain, cold and touch have characteristic position, fibers and ends", as further experience has shown that fixed points – associated, according to Woollard's experiments, with pain, cold and touch reception – are otherwise irritable, or induce different sensation upon stimulation, under changed conditions (fatigue, illness: Droogleever Fortuyn, p. 572).

In addition to giving rise to inadequate sensations by wrongly or variously striking receptors or transferring to the peripheral system, stimuli may produce this effect through

Re 2. Changes in the transmission of stimuli. The peripheral, retrogangliar and central nervous system consists of thick myelinated and thin unmyelinated or poorly myelinated fibres, in the central nervous system there is seen to be a rough grading of thick and thin fibres. Verhaart detected an admixture of fibres of a different thickness in pathways which were thought to be built up of only one kind. Thick fibres conduct brief, fast currents (about 90 M/s) of high potential differences, whereas thin fibres conduct longer, slow (about 2 M/s), low potential differences.

The simplest postulate, to the effect that thin and thick fibres carry pain and touch stimuli respectively, appears to be untenable. On anatomical grounds,

Weddell, (p. 167) speaks of two kinds of pain fibres, thus: "The cutaneous innervation consists of nerve nets subserving probably fast pain and apart nerve nets subserving probably slow pain".
The following chart is Gasser's (see Granit, p. 46):

Cold and heat	thin fibres.
Slow pain	(burning, scraping, crushing)
	(second pain: thin fibres)
	conducting at rates below 2 M/sec.
Fast pain	(prick pain; initial pain)
	(first pain: larger fibres)
	conducting at 15-20 M/s.
Touch, slow	(very) light stroke
	larger fibres: conducting at 2-20 M/s.
fast	hard stroke
	larger fibres: conducting at 90 M/s.

According to Gasser and Granit (see Granit, p. 46): "The fibers belonging to different modalities must be widely distributed throughout the various fiber sizes". It appears, moreover, that "specific sensitivities often were represented in afferent fibers of different size" (Granit, p. 45), while the same writer thinks there are good reasons for answering the following question in the affirmative: "Have we any evidence for the possibility that one fiber can transmit several messages at the same time?" (p. 287).

In accordance with indications in the literature, Granit found the same conditions prevailing in the peripheral, retroganglíar and central nervous systems. The impulses from peripheral stimuli are converted in the receptors into a frequency code (time, velocity, intensity, Granit, p. 50) and are transmitted by a variety of fibres to the central nervous system for interpretation. Hence a stimulus is determined peripherally (in spikes and waves) and integrated centrally for interpretation.

The terms in which this diversity of concepts is expressed are used and understood in different ways.

Interpretation. This means translation or explanation. We should understand somatic, central or cerebral interpretation to mean the conversion – translation – of a coded impulse arriving in the central nervous system into a sufficiently modified impulse for psychical interpretation (= perception) to be possible, irrespective of whether this perception does or does not take place.

It cannot be supposed that an impulse as received in the central nervous system from a receptor could be instantly perceived, without modification, during its passage from one to another higher cerebral centre. The somatic interpretation, which is the prelude to the perception, at the junction of soma

and psyche might be called "the transformation of biological energy into a conscious content by the activity of centres and circuit systems". How this happens and where (thalamus?) is unknown; probably the somatic interpretation synchronizes with the psychic interpretations (perception). It is known from examples that perception may remain in abeyance after a somatic interpretation, from which it follows that the two forms of interpretation are distinct.

Thus a person afflicted with sensory aphasia may hear words and be able to repeat the sounds, without understanding them In cases of astereognosis the sufferer feels the shapes but does not recognise them.

Those who are psychically blind see objects with their eyes – so that they do not run into them – but not with the mind, and therefore do not recognise them.

Psychic interpretation, or briefly, interpretation is the apprehension of the stimulus pattern formed by somatic interpretation and integration.

Integrating means completing, (re-)assembling. As far as the assimilation of stimuli is concerned, this can be done in at least three ways, *viz.*,

(a) Peripheral integration: One fibre carries stimuli from several fibres or receptors.

(b) Central integration: Numerous olfactory and visual fibres stand on one central fibre in the olfactory bulb and in the retina respectively.

(c) Cerebral integration: Numerous impulses are united into one somatic interpretation in the thalamus and cortical projection fields.

For example, the various forms of stimulation which arrive in the thalamus are integrated there into one sensory gestalt. The integration cannot be actualised without the somatic activity of the thalamus. (Hence the integration is determined somatically.) Now it appears that, in the normal individual, at the very moment when integration takes place and this gestalt comes into existence, the integrated functional activity of the then operating senses and nerves *is* experienced at the same time. Man therefore *knows* (experiences) the world around him from moment to moment by experiencing the senses and nerves in action at those moments, *i.e.*, by dint of an obviously synchronous assimilation of the biological stimulant energy from the periphery, a cerebral integration to one sensory gestalt and the experiencing of it.

(d) Psychical integration. This knowledge is integrated with other experiences or reminiscences from the past and is affectively charged with them. It is adapted to the voluntarily chosen or constitutionally determined psychical condition of the person, *e.g.*, toothache is experienced differently by an optimistically inclined individual, who goes to the cinema in spite of it, as compared with the pessimist who is bored.

Not only does the system of thick and thin fibres perform the task of transmitting a determined stimulus as a coded message, but the fibres also activate each other so that (re-)integration to a somatic interpretation (= re-presentation of experience) can be accomplished (Granit, pp. 278 and 287). Thus re-integration, too, starts before the impulses reach the cerebral integration centres.

Through their offshoots, the thick and thin fibres form an internuncial system (in the spinal cord: Verhaart). "Thus, at any time, internuncial effects might alter the nature of the message" (Granit, p. 278). "Undoubtedly integrative and summative processes take place in the cortex and the thalamus. But the study of the literature brings strong support for the assumption that (spatial) summation (of sensory stimuli in man) is founded upon a spinal mechanism!" (Droogleever Fortuyn, p. 571).

Summarising this brief survey and at the same time giving the opinion of the investigator not yet mentioned by name here, *viz.*, Laruelle, it has become evident that highly complicated processes are involved in the transmission of stimuli. It also makes us realise that the neurosurgeon who dares to speak of a "selective section" in a rhizotomy or tractotomy is very bold, to put it mildly.

It appears that, although the transmission-integration apparatus works in obedience to physico-chemical laws, it is at the same time subject in its function to the psyche of the subject and to the effectual operation of other circuits not directly involved in the transmission of pain. The least disorganisation of these systems is liable to lead to inadequate transmission of peripheral stimuli and to inadequate sensations. We quote Droogleever Fortuyn, pp. 572, 573, who, after references to the literature, concludes with these words: "The interaction of elements of localisation and pain demonstrates once more that integration of sensation on higher levels of the nervous system is dependent on correct functioning of both spino-thalamic tract and posterior columns". "Electrophysiology teaches that the 'resting' nervous system is not actually resting. Background activity remains and is sustained for the greater part by the continuous stream of incoming impulses . . . The effect of a stimulus results from the stimulus plus the background activity of the elaborating system. Involvement of posterior roots slows down the background activity of the spinal cord, and in this promotes presumably the painful component of sensation".

These statements underline the fact once again that any departure, however slight, from the normal transmission of stimuli may end in an inadequate perception of the stimulus; and whether one calls these discrepancies "disturbed summation or integration", "discordance" or quite simply "short-cir-

cutting" is neither here nor there. Any pathological condition changes the ground pattern of the physiological conducting of a stimulus. It is not easy to predict the effect of a stimulus within the area of somewhat disturbed conducting tracts and would seem to be neither quantitatively nor qualitatively comparable with the effect of a stimulus under physiological conditions.

Re 3. Inadequate sensations due to disturbed perception. A discussion covering the problems of perception of sensations is beyond the scope of this book. Nor, indeed, would it be within the author's competence. Although an attempt to summarise briefly the opinions of competent authors on how, where and when a sensory stimulus is perceived would likewise be idle, a few possible objections have to be met. After reading the next few pages, we think readers will agree with us that a short discussion is called for.

Testing a person for the perception of sensations is like a game played between the subjectivity of the person being tested and that of the examiner The latter has to "tune in" to the way the subject, using the means at his disposal, reacts to the effect of a known stimulus perceived under the special conditions obtaining for him. The examination turns on representation of a perception and its correct assessment, all other factors are subsidiary.

Do two people looking at a green object see the same shade of green? There are some people who see the same object in different shades of colour if they look at it first with one eye closed and then the other. It is as if a coloured glass were held in front of one eye – green – greener? Yellowish green – blue-green? A lens "displaces" the object in the observer's perception; a sharp-shooter would miss his target, because it *is* not in the place he is aiming at. By tacit agreement we call certain objects "bottle-green", let us say; adaptation has taught us to wear spectacles.

After the application of stimuli that lead to the sensation of pain, the physiologist can measure slow, prolonged currents of action of low potential differences in the nerve. If active currents of the same values (slow, long, low) could be conducted *via* thick fibres (regeneration after crushing? biochemical (strychnine)? psychical??), would the sensation of pain likewise be experienced?

On the other hand, stimuli which usually induce slow, prolonged, low currents of action need not necessarily produce pain. A very young child has to *learn* to *perceive* pain. In infancy this perception is delayed and has not yet come to elicit an unconditioned reflex An infant does not feel "pain" if it is pulled up by the heels or if its buttocks are smacked; it may begin to cry as a protest against being disturbed or from fright. At a later age it comes to associate pinching, pricking and smacking with unpleasurable sensations (warning signals against tissue injury) and with scolding; the sensation of pain is then

bound to an unconditioned reflex, *i.e.*, sensations which, beforehand, were not necessarily painful are now experienced as pain. The same thing can be seen in puppies, kittens or young rabbits, which do not protest, with signs of pain, against being picked up by the scruff of the neck until its unpleasantness has been brought home to them on other grounds.

We might put it this way: A fakir's different attitude to the perception of pain need not rest on a different thalamic make-up. Elimination of conditioned and unconditioned reflexes (reactions) to slow, long, low currents of action in peripheral and central nervous systems, acquired by practice from early youth, enables him to accept laconically the sensations usually associated with them.

Thus the pain reflex is *not* a condition, but a customary consequence of the perception of pain. A patient under the influence of Curare cannot exhibit pain reflexes, but the memory of pain enables him to state that he had suffered pain. Bodily reactions to stimuli which are usually painful provide no evidence of the suffering of pain. Stenvers (p. 1000) described two patients who displayed pain reflexes and had a suffering facial expression but denied experiencing sensations of pain. Again, the patient under shallow narcosis exhibits reactions to pain and even makes the grimaces of a person feeling pain; yet after the operation that person cannot remember having suffered pain.

We are, therefore, also debarred from saying that the lack of remembrance of pain is a critical touchstone for the non-arrival of pain stimuli in a person's cerebrum. The patient who injures his skull while fully conscious undoubtedly felt pain at the moment when his head was concussed, but his retrograde amnesia has also wiped out the memory of that pain.

Action currents, as of pain stimuli, can be electro-physiologically measured in anaesthetized animals; they are presented to the central nervous system for integration, even when the subject is under an anaesthetic. (Electro-encephalographic registration of the evoked potential on the cerebral cortex.) They are not experienced consciously. The fact that pain is unremembered furnishes *no* proof for failure of cerebral integration and cerebro-somatic interpretation. It is horrible to think that, while we are operating on a patient, his central nervous system is being flooded with the resultants of pain impulses.

The central integration of peripheral sensory stimuli which do not culminate in perception in those who are unconscious provides the perfect example of an inadequate (negative) experience. With the return to consciousness, the perception of sensations quickens, these being distorted at first and finally adequate. A person staring into the distance, his thoughts "miles away", may catch the sound of words spoken, and even repeat them, yet the sense of them does not penetrate.

The somatic interpretation of stimuli can be psychically experienced by the conscious individual, the conscious perception being thereby coloured. The adequate perception of a, physically speaking, perfectly assimilated stimulus is, in the last (?) resort, dependent on the characteristics of the psyche. The neurovegetative dislocation of sensitive natures, which may lead to excessive irritability, and the intrapsychic incongruities of an hystero-neurotic, psychopathic and schizophrenic nature are known.

It is becoming plainer why it is that, to take extreme examples, the masochist can delight in a stimulus which is intrinsically unpleasant and the schizophrenic may react to a pain stimulus inadequately by laughing. If asked, the schizophrenic will admit undoubtedly to having experienced pain, but, owing to his "split personality", he cannot experience the sensation emotionally, or is desirous of covering up the effect of the sensation. Similar phenomena occur under physiological conditions. For example, persons who are insensitive to such things will not be able to enjoy stimuli generally experienced as positive (beautiful) as such (adequately), despite the fact that their senses and nervous system assimilated these stimuli somatically to perfection.

There exists a whole range of gradations between physiological and pathological conditioning, *all* of which are psychically determined, whether it be by the constitution, the condition or the free will.

We say it is a fact of experience that causalgia and parapathy occur principally (or only?) among those psychically predisposed to them. To our mind, the leading of a neurotic or psychopathic existence is a condition upon which the possible development of these phenomena depends. Similarly, there is a psychic predisposition to a heightened, or weakened, psychic perception of the sensory stimuli presented for interpretation under the influence of psychological factors which, likewise determined constitutionally or conditionally, are more or less prone to intervene and are liable to vary from moment to moment with the prevailing circumstances; *e.g.*, emotion during the examination.

A psychic predisposition of this kind is a component of the total personality structure of man.

Re 4. Inadequate representation of the fully experienced stimulus. It is not a foregone conclusion that the perception of a stimulus and the associated emotion, if any, will be externalised in a manner for everyone to see. The sensation experienced may be masked from the outside world deliberately, unintentionally, voluntarily or involuntarily (poker face, "I don't wear my heart on my sleeve", stiff upper lip, poverty of affect). The ability to hide one's feelings and the inability to react spontaneously with warmth are also matters of disposition being either innate or cultivated. Similarly, some people are psychically

predisposed to exaggerate their experiences, to represent them inadequately, inappropriately, in a false light in fact; an idiosyncrasy that can be very troublesome when such people give details of their clinical history, especially when it comes to complaints of pain. These characteristics also belong to the total personality structure of man.

As we shall see, *the degenerative back turns out to be* one of the manifestations of a personality structure which, in its totality, may be termed degenerative. One of the difficult tasks we have set ourselves in this study (see p. 413) is to adduce evidence of a consistent association of a degenerative psychical predisposition (also in the matter of perceiving pain) with manifestations of a degenerative back. It is precisely this presumed psychical predisposition to respond inadequately to sensations that makes it so difficult to appraise the results of sensory examinations made upon sufferers from a degenerative back. For, in such a patient we may find, side by side:

Somatic manifestations, *e.g.*, in the spinal column.
Irregularities in the structure of the nervous system.
Irregular functioning of the nervous system.
A psycho-degenerative personality structure.

Therefore, when examining a patient suspected of having a degenerative back, we shall have to allow for the possibilities of false reference of stimuli, inadequate perception of stimuli and false reference of sensations.

Using the concepts defined in the foregoing, we shall once again summarise the analysis of a sensory examination from a physiological and psychological point of view.

The practitioner should bear the following facts in mind when examining a human being for sensory responses:

1. The applied stimulus effects a complicated aggregate of physical, chemical and other changes in the appropriate sense organ. This organ is brought into a state of excitation ("Erregung").
2. The induced excitation is conducted along peripheral nerves into the centre of the spinal cord and brain stem, in which summation, integration, activation may take place. From here, transmission is effected in the central nervous system along afferent projection systems.
3. Perceptions are made thanks to the activity of higher levels of the central nervous system. These perceptions can, in a general way, be divided into cognitive and emotional sensations. Owing to higher integration, which presupposes a given activity of certain parts of the central nervous system,

the affectively charged content of the known enters the conscious at a given moment. Needless to say, there are numerous affectively charged contents which do not enter the conscious of the experiencing subject. They may be qualified as unconscious experiences or unconscious sensations.

The nature of the sensation is primarily determined by the nature of the peripheral stimulus. We know by experience, however, that if different individuals are exposed to the action of a given stimulus of a given nature, they do not by any means experience the same sensation; quite the contrary, each individual seems to have his own, subjective sensation. This shows that the nature of the sensation is very particularly determined by the intrinsic quality of the personality structure, whether hereditary or formed by the individual's historical development. By this we are to understand the total effect of earlier experiences of life, both the unconscious ones (in early childhood) and the conscious experiences. (Education, culture, identification processes and other factors play a part here.) The quality of perception of sensory stimuli is also determined by the phase of life through which the individual is passing. Lastly, when the intellect has been developed, conferring the power of rational evaluation upon the individual, it will contribute its quota to the determination of the quality of the sensation.

Thus, sensation may be disturbed by:

A disordered peripheral sense organ, owing to which the excitation is not initiated.

Disorganised transmission (peripheral and central afferent systems).

Inadequate activity of the cerebral areas.

Hereditarily determined disturbances within the personality structure:
weakness of instincts;
weakness of drives,
impoverished emotional capacity,
temperamental disturbance.

Disturbing unconscious experiences of early childhood

Disturbing unconscious experiences of later life.

Disturbing conscious experiences of life.

Faulty upbringing and instruction.

All disturbing influences which bring about pathological identification.

Intellectual disturbances, including abnormal individuation.

A larger incidence of one or several of the above disturbing factors is to be expected in those persons who can legitimately be expected to have a personality structure which, in its totality, is degenerative, one manifestation of which is the degenerative back. For this reason, in such cases the findings at sensory examination in practice have relatively little value and we therefore consider the assistance of the psychiatrist and the psychologist to be indispensable.

B. Psychic disturbances. Use and misuse of the terms "psychogenic" and "functional"

The result of neural therapy ("Sekundenphenomen"), acupuncture, various manipulations in chiropractice, effects of cocainisation of the spheno-palatal ganglion, the reflectory disturbance in the collateral circulatory system according to Leriche; all these are mysterious happenings not yet properly understood. Like anaesthesia dolorosa, causalgia, parapathy, phantom pain and neuralgia, they are still unexplained phenomena which, in this sense, are of the same order as the residual function phenomenon. The inexplicability offers no excuse least of all to the critical examiner, for writing off the patient without more ado as psychogenically deranged. It is right and proper to consider that the patient's nervous system and psyche may *function differently* from the way we suppose them usually to do. We hesitate to speak of *functional* phenomena, because the word "functional" is often resorted to when an anatomical substrate cannot be found for a certain phenomenon and the practitioner is at a loss for any (other) (exact) explanation for it. Accordingly, the concepts "functional", "psychogenic" and "neurotic" are used indiscriminately and are often interchanged. So that we may be properly understood, let us first attempt to define what *we* mean by these terms.

When we speak of a functional phenomenon, we wish to imply that these particular signs and symptoms derive from changes in the function (activities) of tissues, organs or organic systems. Then, if one wishes to define the functional more exactly, a further distinction should be made between the organically functional and the psychically functional.

I. If we say that an established phenomenon is *organically functional*, it means that organic changes in the structure of tissue, organ or organic system are responsible for the functional changes just referred to. Hence organically functional and organically caused, or better still, organically conditioned, are identical.

II. We call a phenomenon *psychically functional* (= psychogenic) if the detected change in function is conditioned, not by changes in organic structure, but by psychological or psychic factors.

Although psychically functional and psychogenic are synonymous, psychogenic and neurotic are not. For instance, non-neurotic fear can bring about functional changes in tissue, etc. The change in activity which can be detected at that moment is then caused (psychogenically or psychologically) by fear. From this example it will be clear that psychogenic is more comprehensive than neurotic.

Neuroticism is a certain aspect of the psychogenic. We say that a symptom is neurotic if the following conditions have been satisfied:

1. Neuroticising factors must have come into action which are experienced by the individual as psychically unacceptable.
2. These neurotic factors must put the brake on the development of the individual so that a fixation supervenes, or a regression to the individual's levels of development in early childhood.
3. The most conspicuous of these developmental disturbances is that of the individual's sensuality and sexuality, or, in psycho-analytical terms: the development of the libido.
4. If neurotic development is in question, it should be possible to discover repressive dynamisms in the patient; that is to say, there should be a repressive influence somewhere, usually a repressive affect, found generally to be fear expressing a repressed content.
5. The neurotic individual is not conscious of the repressed content.
6. In spite of the repression, the neurotic content tends to be realised on a lower level (sublimation, symbolisation and other neurotic dynamisms).
7. The neurotic with a split personality structure lacks to some extent the optimum integration between his "I" and his "Es".
8. Generally speaking, recovery from severe neuroses is not spontaneous and can only be accomplished by way of psycho-therapy.

III. In addition to the organically and psychically functional, there is a third category of functional abnormalities which are conditioned constitutionally (labile condition), in association with which the endogenous disposition lays the individual open to episodic (cyclic) extrapsychic disturbances of the working economy of his organism. This is to be understood as *functional within the framework of disorganisation*, the disorganisation being as a rule conditioned by the genotypical constitution. We could therefore call it "disruptively functional". As we see it, however, there can be no doubt that disruptive functional abnormalities can also be acquired by unknown factors in the course of a lifetime, in the absence of any organic or psychic influence. We are thinking more especially of cosmic-tellural influences ("rays", full moon), endocrine influences and the like.

IV. *Constitutionally functional.* Finally, there is a fourth category of functional abnormalities, involving activities conditioned neither organically nor psychically in the ordinary sense and which cannot legitimately be described as disruptively functional disturbances. They should be considered as the expression of an abnormal receptivity of the nervous system, conditioned by the genotypical constitution. This is where we come upon the problem of the genotypically determined abnormal temperament, this latter being a special factor in the personality structure. The temperament is known to determine the formal manner in which the tissue, organs or organic systems (hence also the nervous system) act(s) and react(s); ultimately, therefore, the formal manner of the whole personality. By this is meant the qualitative and quanti-

tative receptivity of the individual which impresses his prototypal stamp upon him.

Re 1. It falls within the framework of this study to divide the organically functional changes into two groups, *viz.*,

a. Organically conditioned changes of function in the strict sense as the result of known changes in organic structure. This does not call for explanation.

b. Functional changes, however, also occur which, for want of a more euphonious term, we shall have to describe as "pseudo-psychogenically organically functional". The causal or conditioning changes in organic structure responsible for these functional changes are not known in the case under examination.

This second group has been *formed on theoretical grounds, viz.*, that, on the analogy of identical cases, there is possibly an anatomical substrate – which, indeed, is sometimes found – and that there is insufficient evidence for qualifying the functional change as a psychogenic one.

This clumsy term "pseudo-psychogenically organically functional" therefore stands for functioning "differently" as the result of an actually unusual structure of some organ (*e.g.* the nervous system), even if this different structure is unknown or undetected. It may seem supererogatory to mention this possibility, but practical experience has shown that it is not

A structure of this kind may be endogenous (uncrossed pyramidal tract) or the disposition may predispose to an *unusual function of the nervous system following an* event which does not generally set off this reaction (parapathy, causalgia). The dissociated sensory disturbance behind a severed posterior root furnishes an example of an exogenous (pseudo-psychogenic) *organically functional disturbance.* (See p 365). Now we do know that this disturbance is not psychogenic, but so far we have no satisfactory explanation for this dissociation

Neuralgia, notably neuralgia major of the trigeminal nerve, proffers an excellent example of this group, provided we accept the definition of neuralgia as being a state of irritation in the innervation area of a peripheral nerve without any known substrate. Up to the present the cause of neuralgia major has not been convincingly demonstrated, arteriosclerosis has been rejected as the only explanation; Taarnhoj's postulate that the nerve is pinched between capsule and tentorium has been called in question Should, however, the aetiology of neuralgia major become established beyond doubt, there would be, *ipso facto*, a reason for calling it neuralgia minor. Even *if the substrate remains unknown*, we may still legitimately call trigeminal neuralgia an organically functional disorder, probably the subject is predisposed to it, its occurrence in families is known

A presumed varying structure of the nervous system *is likely to remain* uncorroborated, it will seldom be demonstrable at post mortem and only most exceptionally through surgical intervention This applies to gross anatomical variations; finer structural anomalies are seen microscopically, though there is *no verified association* with the function (*e g*, the fibre structure of the pyramidal tract and of the spino-

thalamic tract according to Verhaart). The literature provides us with no definite information on anomalous functions of fibres or cells.

On one occasion we were able to show by chordotomy followed by minute examination that the spino-thalamic tract was uncrossed, *i.e.*, that the protopathic transmission of stimuli did not cross in the spinal cord. Before this diagnosis was made, the patient's symptoms were held to be psychogenic, whereas they were in fact organically functional.

After unilateral chordotomy the patient sometimes passes through a transient condition of weakness in the homolateral leg, generally without a hypertonic concomitant and without increased or [illegible]. this kind has on several occasions [illegible] supposition may not always have been groundless, since a patient with tendencies towards psychogenic reactions, on becoming aware of the analgesia in the homolateral leg, identifies this with, or converts it to, a paresis

That great care has to be exercised in the drawing of conclusions will appear from work done by Nathan and Smith with reference to the correlation of the plantar response with *proved* lesions of the spinal cord after antero-lateral chordotomy. We quote the following from Spiegel's summary of this (1957, p 163): "Any lesion of the lateral and ventral columns of the cord may or may not cause the Babinski response". "The Babinski response is often found with a histologically normal cortico-spinal tract". "In sum, there is no particular relation between the anatomic state of the cortico-spinal tracts and the form of the plantar response. The Babinski response is pathologic; it may be taken as an indicator that there is an abnormality of function in the central nervous system".

Organically functional syndromes can be influenced by surgical intervention in the nervous system (contralateral chordotomy, sympathectomy for causalgia, peripheral nerve treatment for neuralgia), even if the structure of the nervous system is known to be unusual (homolateral chordotomy). The result is not always the same, owing to:

(1) erratic anatomic relations,
(2) difficulty of diagnosing and formulating indications,
(3) the admixture of psychogenic components which make the prognosis unfavourable

Psychogenic components in the syndrome will make a correct interpretation difficult, because, in the event of an abnormal anatomy of which the examiner is not aware, certain symptoms will seem inexplicable to him. Moreover, in our opinion, a predisposition to unusual organic functioning – due to the peculiar anatomy – is linked with a predisposition to psychical disturbances.

In view of the above considerations we should say that "psychogenic disturbance" is an unwarrantable diagnosis if based on the inexplicability of symptoms or the fact that some symptom does not fit into the defined neuro-

logical picture. Such a diagnosis demands primarily a positive symptomatology on psychic/psychological grounds; then and then only can symptoms that are plausibly conversions complete the psychiatric picture. From this it follows that psychiatric/psychological comprehension forms the background of every diagnosis and for neurological diagnosis especially. Hence neurological and psychiatric examination is indivisible (neuro-psychiatry), whether the examiner possesses enough psychiatric experience to deal with an uncomplicated case, or, on principle, calls in a psychiatrist. It is our personal opinion that no surgery should be performed to combat subjective complaints – least of all so-called pain surgery – without previous psychiatric examination.

Re II. The psychically functional symptoms may be conditioned, but are not caused by changes in organic structure. Consequently, it is an established fact, by definition, that an anatomical substrate cannot be the cause of the changed function. This notwithstanding, the psychogenic symptoms can be divided into two kinds, *viz.*, those which do and those which do not involve the organism.

a. In the purely psychically functional it may be assumed that psychological or psychic factors cause the nervous system to function in the usual way, yet so that the result of the function is different from the ordinary reaction to an agent (a pin-prick is said not to be painful).

b. Pseudo-organic psychically functional disturbances obtain this anomalous result by causing the nervous system to function in a different way organically, without any alteration to its structure.

This possibility is taken into account in psychiatry and the psychical disturbance is said to be the [illegible]

Fo [illegible]

wish [illegible]

(pain) may either be [illegible]), or actually be [illegible] of affairs could be [illegible] nerve tracts, *i.e.*, [illegible] ermed psychically [illegible] by changes in the nerve cells and be measurable by, say, potential differences In that case we should be confronted, not with the "substrate" of a psychogenic disturbance, but with its secondary manifestation

Let us not too hastily suppose that this is an impossibility. Not very long ago it was postulated that a peripheral lesion (section of an afferent nerve) does not change the central nervous system. It is, however, a known fact that the pathologist reports ascending degeneration in the spinal cord behind a severed root; pycnotic changes in the nuclei of the cells in the spinal cord are noted in parts of the lateral geniculate ganglion and in the cortical projection fields of a blind eye. The activity of a part of the body can, under certain circumstances, be registered electro-encephalographically, the absence of an extremity, *i e*, the absence of a background activity, is measurable (Bremer, see Droogleever Fortuyn, p. 573); a general slowing down of electrical activity of the spinal cord follows section of posterior roots (Ten Cate *et al.*, see ditto)

Similarly, we are persuaded that a sensory 'blind spot" is formed in the central

nervous system behind a severed peripheral afferent fibre and that the functional, or anatomical *and* functional, changes will therefore prove to be demonstrable in cells or pathways of the central nervous system

For the same reasons we do not think it is impossible that chronically pathological function of nerve tissue might eventually produce perceptible secondary changes in the central nervous system

In the foregoing we have submitted a conceptual definition of the term "functional", have taken our stand against the careless use of this term and explained why a too ready resort to the word "psychogenic" is to be deplored. Briefly, the word "functional" does not signify "untrue", or "false", to the detriment of the patient, and should certainly not be bandied about in that sense if we mean "inexplicable", or "not yet known", which is the more honest and the more honourable of the two, because it implies that it is *we* who do not know.

II. Relationship between morphology and function

It will be convenient to deal with the following subjects separately.

The mechanism of referred pain.

In a comprehensive survey of the literature, Sinclair, Weddell and Feindel (p. 184) relate the opinions there expressed as to *how* referred pain and referred tenderness are induced.

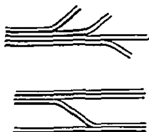
"Stated broadly, then, the numerous shades of opinion can be reduced to two opposing beliefs. The first of these is that some (misinterpretation) mechanism located in the central nervous system is responsible for all the phenomena of referred pain, and the second is that these phenomena are produced by events taking place in the periphery".

"Nevertheless, the fact remains that, as they are at present stated (1948), neither hypothesis is capable of explaining all the observed facts". They submit, as against these two hypotheses, their own postulate "that axon branching is responsible, at least in part, for the occurrence of referred pain and its associated phenomena". "This branching is of such a type that one limb of a branched axon passes to the site of origin of the disturbance and others pass to the sites to which the pain is referred. This mechanism works in two ways; first by leading to a misinterpretation by the central nervous system of the true origin of the pain impulses, and secondly by the liberation of metabolites at the terminals in the region where pain is experienced, thus giving rise to secondary pain impulses having origin at the periphery".

The tenability of this theory, however, depends upon whether axon branching does in fact occur. In the main, the facts do not indicate that it does.

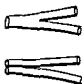
Referred pain has proved to be inducible in phantom limbs (Harman, see Sinclair); therefore a peripheral apparatus (axon branching) is not a requisite.

Bifurcation of the axon was observed histologically in the frog and in mammals and electrophysiological evidence of occasional axon branching is known in the frog (see Sinclair *et al.* p. 193); but, as far as we could discover from the literature up to 1958, there is no evidence that this particular arrangement also occurs in man. Moreover, axon branching was so seldom demonstrable in animals that, even if there were reason to assume by analogy that the same phenomenon occurs in man, its occurrence is too infrequent to account for referred pain.



Axon branching is compared to the terminal fine branches of a nerve (Weddell). It remains very much to be seen whether the terminal division of nerves is effected by the formation of fine side branches or by the separation of small fibres. The latter is in keeping with the ground pattern of the peripheral nerve, which becomes steadily thinner as it gives off fibres towards the periphery. On rare occasions fibres jump over to other nerve bundles, but without merging with their fibres.

It seems inconsistent to attribute undemonstrable properties to the terminal branches which the anatomically dissectable parts of a nerve have been shown not to possess. In view of the abundance of nerve tissue with which the body is provided, it seems equally inconsistent to suppose that various tissues (skin, muscle, viscera) remote from each other should be innervated by one and the same fibre, even if they correspond segmentally.



The branching of a slightly larger nerve fibre has been proved neither anatomically nor microscopically. The comparison with current conducting by two wires soldered together is not valid.

Sinclair *et al.*, reviewing the literature up to 1948, were unable to adduce proofs of the existence of axon branching in man; we have not come across either descriptions or drawings of axon branching in the subsequent literature. In modern physiology, on the other hand, the existence of an *integrating*, summational, internuncial system has been becoming ever more evident. Even Sturge (1883), Sherrington (1892) and Mackenzie (1909) were convinced that a spinal mechanism might be involved. "Bahnung", timing, integration,

summation, activation – all these are attributed to the function of a complicated internuncial system (Granit, Walshe, Droogleever Fortuyn, Verhaart and others). The anatomical foundation for this view is the demonstrability of "reflex collaterals from any given dorsal root, ramifying over a number of neighbouring segments" (of the spinal cord). (LLoyd, see Sinclair *et al.*, p. 187. Cajal, see Droogleever Fortuyn, p. 567, Ranson, Szentagothai, see ditto). Lopez Cardozo drew attention to the intersegmental extension of afferent fibres, which is part of the anatomical basis for the function of transitional fibres assumed to exist on other grounds and described by us previously.

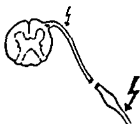
We purpose to submit that the existence of transitional fibres does not conflict with known physiological data, in this case respecting referred pain. Now, as explained and in accordance with the opinion of many, we may ascribe referred pain (with analogous phenomena) to a "misinterpretation mechanism" of the internuncial system in the spinal cord. Therefore, the peripheral projection of transitional fibres can be excluded from the problem of referred pain, although the variability of the innervation patterns of transitional fibres as well as their intersegmental extension in the spinal cord might well help referred pain to pass beyond the confines of the segmental We have already spoken about the associated sensitivity to stimuli of nerve endings in the innervation areas of roots adjacent to an injured or diseased root.

Summation of peripheral stimuli on a "central focus".

Quoting Droogleever Fortuyn (p. 573), we repeat: "The effect of a stimulus results from the stimulus plus the background activity of the elaborating system".

An anatomico-physiological explanation of background activity as an undercurrent for pain stimuli below the stimulus threshold depends on the "coincidence of the cortical pain projection centre in the thalamus with the epicritical projection centre" (Ariens Kappers, p. 805, Foerster, see ditto) In other words, the conductors of both qualities of sensation lie close together in the thalamus, so that purely epicritical stimuli (a gentle caress, perception of light or a noise) may produce conscious sensations of pain in hyperalgesic subjects (as, for instance, is known to be the case with people suffering from trigeminal neuralgia). In a patient suffering from this form of neuralgia a tactile stimulus results in an attack of severe pain. Peripheral interruption of the background activity (infiltration by alcohol, neurexeiresis or retroganglar section) prevents attacks of pain. The central integration and perception apparatus remains unchanged, as far as we know, yet the cause of neuralgia major is considered to reside in this as the result of disturbed functioning.

Attacks can be kept at bay for varying periods by a violent discharge of this apparatus (manipulations to the nerve without section) in the conscious or unconscious patient; several examples are to be found in the literature, including Dandy, Hanraets (1954). As the result of the elimination of the background activity or the temporary interruption of a disturbed cerebral circuit, stimuli from a central focus are not perceived.



Sensory rhizotomy in a caudal root will, in appropriate cases, banish pain previously localised in the corresponding root area. From this, neurologists practising pain surgery conclude, often rightly, that the cause of the pain attacked the root at a point peripheral to the site of section, or the peripheral nerve. Actually, however, this need not necessarily be so (Weber and Fechner's law). A more centrally situated focus (spinal cord or root closely adjacent to it) can excite pain stimuli which, themselves, remain below the sensorily perceptible threshold of pain. The continuous stream of incoming impulses from the peripheral nervous system may increase the total quantity of impulses to such a pitch that, carried along by this stream, the pain stimuli pass beyond the threshold. The interruption of this stream (rhizotomy) may prevent these centrally initiated pain stimuli from being perceived (medullary tumour, caudal tumour).

One asks oneself with amazement why a stimulus like that formed by the compression of a root by a hernia does not *continuously* produce sensations of pain. The proposition that the root has become emasculated by degeneration does not furnish the only explanation, seeing that degeneration has by no means always been found (*e.g.* after extirpation in rhizotomy), while the motor and sensory functions may remain intact. Some patients suffer pain continuously from the compression of a root, *even when at rest*: the pain stimulus is situated above the threshold of pain. The majority, however, experience pain during movement, *i.e.*, action accompanied by the initiation of a greater influx of efferent-afferent action currents through the root involved. We do not know whether increased pain on movement is due to a quantitative increase in mechanical pain stimuli, or also — and perhaps mainly — to the emergence of stimuli, otherwise kept below the threshold of pain by root compression, which are carried beyond that threshold by increasing background activity.

It could be countered, rightly, that only certain movements are painful, adding that these are the movements which produce enhanced activity in the innervation area of the compressed root.

Extreme movements, comparable to those performed during the Lasègue

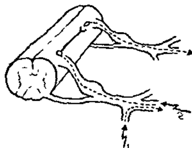
test, by which a mechanical factor is activated, might seem to be alien to the argument, but on closer consideration they will be seen to reinforce it; for, in serious cases, the Lasègue only becomes positive at 30°, not infrequently only at 45° or 60°; Charnley saw that the roots do not move as long as the legs move, or are moved, at an angle of less than 30° to 40°. Now, a patient who experiences pain when moving does not walk with straight legs in a stooping posture. He tries to adjust his motoricity so as to avoid pain, and fails in this attempt under certain circumstances, even if the amplitude of his movement is so small as to make displacement of the roots improbable.

This perhaps explains why the patient with chronic root compression ceases to have radicular pain – despite the absence of sensory disturbances – when the corresponding musculature is atrophic.

How else could radicular pain be brought about in association with movements too mild to cause the root to slip over the hernia? From the mechanical point of view, a varying degree of protrusion at each step is unthinkable (bud hernia, hernia in lateral recess, hernia in foramen or intervertebral canal, detached hernia). Yet the patients suffer pain when moving under circumstances in which neither the hernia can change its position, nor the root in relation to the hernia.

Distant referred pain. The peripheral focus.

A single, brief stimulus can have a prolonged after-effect, even though the referred pain thus induced is of different modality. Muscle tension and vascular reactions may attend this pain, or occur independently; biochemical reactions at the terminal extremities of the nerve are thought to take place. A biochemical reaction as the result of the first stimulus ("trigger point") can subsequently serve as the stimulus in a following circuit (Wolfe and Hardy, see Sinclair *et al.*, p. 206). Thus a chain reaction is set going, through which the stimulus may migrate, from the viscera, say, *via* muscles to the skin, segmental boundaries thereby being passed. The hypothetical axon branching might be involved in the formation of a circuit, or circuits; but the migration of the stimulus is suggestive rather of efferent-afferent reflexes, while intersegmental extension for some distances requires activation of the internuncial system. "As Theobald (see ditto, p. 206), however, points out, if the central nervous system works in such a manner as to permit of the spread of 'excitation' over the distances required



by this theory, there would result a condition of chaos, both in the spinal cord and in the brain. It may be noted, in passing, that a similar explanation may well apply to the results of anaesthetizing painful nodules in a skeletal muscle. Anaesthesia of most of these produces no more than a local result, but occasionally there is found a 'trigger point' (Livingstone, 1943), the anaesthetization of which leads to the abolition of pain and tenderness throughout a wide area. It is quite possible that this 'trigger point' represents the original source of impulses, which have set up secondary sources, each of which may have produced tertiary sources, and so on".

The thoughts embodied in this quotation offer a scientifically grounded explanation of Huneke's "Sekundenphenomen", which is why we quote it here. We wish to state at the same time that we have seen this phenomenon induced and have ourselves succeeded in inducing it.

The theory states that anaesthetization (Impletol) of a peripheral focus (trigger point) also abolishes distress elsewhere in the body (distant referred pain) within a few seconds. Huneke ascribes this to the interruption of a deranged balance in a circuit built up by the peripheral focus (field of disturbance), this circuit invading territory beyond the anatomical boundaries.

It is not difficult to see points of similarity between the two views and the events to which they refer. While we are personally of opinion that the Sekundenphenomen can only infrequently be induced and that the patients upon whom neural therapy can be tried out have to be carefully selected (see p. 578), we nevertheless accept, with Huneke, the existence of the phenomenon and think that a scientific explanation for it may well be found.

Neural therapy is somehow connected with the "neural pathology" of Speransky. Like many other research workers, (Sanarelli, Scheidt, Spiesz), Speransky regards the nervous system as the centre of the organism regulating everything; he holds that disturbances of it are the cause (or the condition? – present author's query) of all pathological processes, including infectious diseases. The nervous system is supposed to consist of many electro-biochemical circuits, each cell being surrounded by fibres of an exceedingly fine network, in comparison with which the currently known physiological and anatomical particulars constitute merely a gross representation. A preceding influence or disturbance is retained in this neuro-biochemical somatic pattern and conditions the effect of a subsequent influence or disturbance. Neurodystrophic processes are initiated in this way, which might be pathogenic. Let us illustrate this with an example of our own: An electric motor revolves to the left or to the right, according to the coupling of the wires. If the motor is already turning, started by hand for example, the switching on of the current for a few seconds results either in acceleration or retardation of the existing motion. If we insert the adjective "pathological" before "acceleration" (burning through, running hot), these few simple words clarify the basic idea of conditioning by neural pathology. We are, however, far from knowing how deranged circuits are to be influenced therapeutically,

that is to say, whether positively or negatively. On the whole, neural therapy appears to resort to negative action (anaesthetization), while other neuro-therapeutic interventions and acupuncture would seem, by the inducement of (pain) stimuli, to tend more towards the positive. Hence the surprising contradiction between these two methods of treatment is only apparent.

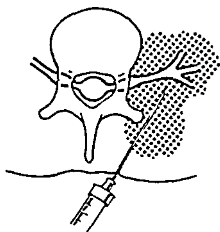
The theories on neural pathology and the practical application of neural therapy have been resisted in the past and still are today, in spite of the fact that successful experiments, which we shall not discuss in detail here, and promising results support these theories. It is the author's personal opinion that this resistance was engendered by the fact that these somewhat mysterious-sounding theories appealed most to and were applied by those exponents of the idea who were unable to distinguish clearly between cause and condition, unlike the founders of the theories, *i.e.*, Speransky and others The neural therapist who represents relief of pain or symptoms as a cure, in the sense of removing the disorder or disease, cannot be taken seriously by academic science, because he uses unacademic scientific terminology. But, neural pathology, viewed as a conditioning for the action of exogenous factors, and neural therapy, as the palliative influencing of a condition, are not revolutionary. The one is a different point of view, probably largely correct, while the other is a different, sometimes valuable, form of therapy.

The effect of anaesthetizing the area of reference – in this case "neural therapy" – has been known for some considerable time (since 1926) in academic medical literature (see Sinclair *et al.* p. 203). The fact that anaesthetization of a peripheral area successfully combated referred pain was advanced as a counter-argument against the view that referred pain was generated *via* a central mechanism in the internuncial system (Davis and Pollock, Ariens Kappers, p. 787). It was found difficult to refute this counter-argument on the basis of the available information in the literature.

It seems to us that the following suggestion should be considered. Background activity constitutes the undercurrent on which stimuli below the stimulus threshold nevertheless pass this threshold. If the primary impulse (to referred pain) remains below the threshold on its arrival in the internuncial system, it needs the undercurrent of background activity to get over the threshold. This undercurrent is cut off when the area of reference is anaesthetized, consequently, the primary stimulus is not perceived. If, however, the primary stimulus is of sufficient intensity to pass the threshold on its own power, anaesthetization of the area of reference will not abolish the referred pain, or will merely soothe it. (This is a known phenomenon in practice.) This suggestion refutes the counter-argument and at the same time explains why neural therapy so often fails

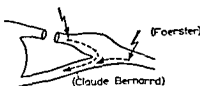
Anaesthetization of a root where it is compressed, say, by a hernia, temporarily eases the pain. This is not astonishing At first, we were greatly impressed when we saw how, by paravertebral anaesthetization (peripheral nerve at the

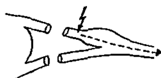
exit of the intervertebral canal or the plexus), a patient who, as was later verified at operation, had a radicular syndrome due to *root* compression by a hernia was temporarily relieved of pain. The patient was also able to move his back and limbs without pain. The phenomenon, which is not always positive, is a known neurotherapeutic treatment. It must be assumed, to account for this, that the anaesthesia shut off the background activity, supported by which pain stimuli below the pain threshold had previously been perceived in the more centrally situated root.



Antidromic conduction of impulses.

The phenomenon is well known, *viz.*, the stimulation of a peripheral stump of a severed nerve, if sufficiently intense, produces the sensation of pain. Many as are the theories that have been advanced, the fundamentals of this phenomenon are not known. One thing, however, is certain and it is that, to materialise, the peripheral impulse must reach the central nervous system *via* nerve tissue which is in contact with it. Conduction *via* afferent pathways into the motor root has been demonstrated (Foerster), but must be ruled out as the only possible explanation in this case, because the pain stimulus penetrated even after section of the motor roots (Foerster). Many (Foerster, p. 307) think the explanation resides in transmission *via* sympathetic distant plexus. It is true that sympathetic fibres are able to conduct pain (Granit, p. 110), but Sturup and Carmichael (p. 216) proved experimentally that "in man the periarterial plexus does not serve as an afferent pathway for pain from the periphery".





This opinion is expressed with the proviso that sympathetic fibres are undoubtedly able to conduct pain, but they communicate with the spinal cord *via* the roots, but the periarterial plexus would be incapable of bridging the distance which a peripheral stimulus would have to travel from an area below the transverse lesion (or several injured roots) to posterior roots above the lesion

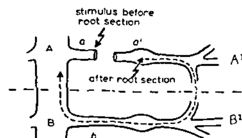
Antidromic conduction to the central nervous system is said to be due to a

short circuit in the terminal branches of adjacent peripheral nerves. According to Weddell (p. 170) and Granit (p. 43), however, "The terminal ramifications *of every main fiber . . . were found to remain independent from those of other* fibers". Hence the supposed short-circuiting must take place through other mechanisms. True, pre-terminal anastomoses are described anatomically; also, the merging of a fibre of one nerve into another nerve has proved to be physiologically demonstrable, but the merging of fibre into fibre (or axon branching) in human beings has not been demonstrated microscopically.

We are left with two alternatives, *viz.*, either the transmission of a stimulus through a true short circuit between two proximate naked fibres, or the setting-off of a biochemical process at the extremity of a terminal branch, this process serving as a fresh stimulus for afferent impulses to the spinal cord (Penfield, Davis and Pollock, Lewis, see Sinclair *et al.* p. 188).

Either view *falls in with the interpretation of transitional fibres.*

In effect, a preterminal anastomosis is a peripheral transitional fibre which at the last moment joins up with the fibres of its own segment. If electrophysiological short-circuiting between two naked peripheral end-branches is possible (see Lindblom and Rexed, p. 431) by injury to the nervous structures, then there is no reason whatever for excluding transitional fibres. Intense stimulation of a peripheral nerve stump and, as has been actually observed, a peripheral sensory *root* stump, is experienced in the same innervation area as that of the previously intact nerve or root! Not, however, in the innervation area of the adjacent root, through which, after all, the stimulus is returned, although there is a certain amount of spreading. Afferent impulses coming in through the root (b) should be perceived as belonging to segment B and coming from the skin area B^1, *if fibres from* B^1 *overlap* A^1 *in accordance with Foerster's classical hypothesis.* If it is a fact that stimulation of the peripheral root stump (a^1) starts off a biochemical process in skin area A^1, which in turn serves as a stimulus for impulses which nevertheless arrive in "segment" A of the spinal column *via* root b and are thus experienced as coming from A, *then this is an argument in favour of the existence of transitional fibres.* For, although stimulation of root a does not determine the dermatome (root a also contains fibres from segment B), stimulation of the peripheral root stump a^1 is the best way of exploring the root area belonging to segment A; by the antecedent



section of root *a* it is possible to discover a connection between skin area A[1] and "segment" A of the spinal cord *via* the fibres in a neighbouring *b* root. We called these transitional fibres.

Overlap of the sensory modalities of feeling

As in several previous chapters, we have made it our task to discover the extent to which known physiological facts correlate with our practical experience. In this pursuit we are struck again and again by the paucity of verified facts in this field and by the frequency with which authors of repute differ in opinion on fundamental matters.

Ariëns Kappers (p. 789) holds that pressure on a nerve first cuts off the thick fibres and that cocainisation first paralyses the thin fibres This is called in question by Granit (p. 46), to put it mildly.

Followers of Head repeated his experiments (sensory disturbance and re-innervation after section of peripheral nerves), some with the same results and others with deviating ones (see Walshe), yet as far back as 1937 Ariëns Kappers (p. 790) pointed out that one peripheral nerve contains many, while another contains few thick or thin fibres and that the sensory disturbances resulting from their severance are accordingly of a preponderant modality.

Neither in the literature (*e.g.*, Klein, "Die sensible Chronaxie"), nor in practice (*e.g.*, by comparison with the disturbances after rhizotomies) were we able to find any support for Biemond's opinion (1956, p. 18) that the lesions of a root and of a peripheral nerve are distinguishable by a difference in loss of sensory modalities.

We found insufficient attention paid in the literature to important data such as appear in Biemond, p. 17 and Droogleever Fortuyn; the absence of overlap over the axial lines of the extremities, and, in Biemond, pp. 18 and 236. distal segmental parts and ventral rump areas are disturbed sooner and in a more pronounced degree than proximal parts and dorsal areas (*loc. cit. p.* 373).

The area behind a severed root cannot be reinnervated by regeneration of the retroganglial fibres involved. As has already been stated, this reinnervation is effected by.

(1) Through-growth of fibres from adjacent structures (the result of overlap according to Foerster).
(2) *Via* the anterior roots (= residual pathway, Foerster).
(3) Transitional fibres (= residual function).
(4) S[illegible] s(?).

[illegible] return after varying intervals, *i.e.*, first the [illegible] of pain, burning sensations and hyper-

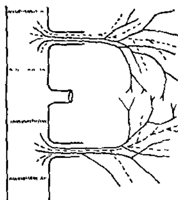
sensitiveness; then the gross sense of touch and finally the delicate sense of touch and of pain. The failure to distinguish between these two different modalities of painful sensation caused confusion at first and is probably in part responsible for the non-correlation of certain experiments.

Let us now, with reference to these summarised facts, enquire what happens *after the section of a sensory root, so as to be able to compare our findings* with practical experience after rhizotomies and with the compression of a root (*e.g.*, by a hernia). Foerster showed that no sensory disturbances occurred after section of one posterior root, or else only a slight disturbance of the sense of temperature and pain, or again (seldom) a disturbance in the sensation of temperature, pain and minor disturbance in the sensation of touch. He makes the following statement, which we quote (p. 285): "... sind auch beim Menschen die Tastdermatome grosser als die Schmerzdermatome". Foerster's opinion is generally accepted, with the exception of Biemond, p. 17, who says that it is a significant fact that the dermatome for the sensation of pain is still larger than that for the tactile sense.

Our observations after monoradicular selective sensory rhizotomies of roots L.4, L.5 or S.1 can be briefly summarised as follows:

No or little disturbance of the tactile sense.
Hypalgesia.
Thermanaesthesia

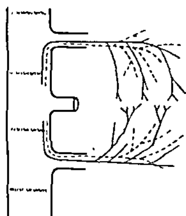
How are we to account for this dissociated sensory disturbance? And what significant inferences can we draw from it for practical work, notably in the case of the partly injured or compressed root? It seemed obvious that the explanation would be found in the *overlapping* of the peripheral sensory innervation and that the dissociation could more especially be related to the opinion held by Foerster, *viz.*, that the "dermatome of touch" is larger (or seems larger) than the pain "dermatome" ("dermatome" = root area). For it follows from this that the larger tactile dermatomes of two neighbouring roots may extend sufficiently to overlap the denervated area behind the severed root, whereas the smaller dermatomes of pain are not able to do so. In that case a hypalgesic or analgesic area will be formed between the two intact roots, and that correlates with the findings in practice The same applies to other modalities of sensation, in the sense that the thermaesthesia dermatomes, for instance, are smaller still.



Pain . . . .; Touch ——

This explanation seems plausible. The discovery of a dissociated sensory disturbance behind a severed root would not seem to conflict with the classical view of overlapping dermatomes as expounded by Foerster. But then one might expect in a denervated area of that kind, where only tactile stimuli are still perceived, that the undegenerated fibres are predominantly thick, myelinated fibres; in other words, that the local, thin, unmyelinated fibres will degenerate massively, whereas a small number of thick fibres (from the neighbouring roots) do not degenerate. This, to our mind, has not been shown to be the case (see Ariëns Kappers, p. 786).

It is almost inconceivable that a convincing finding of that nature should have persistently escaped notice. To us it seems improbable, to say the least, that a phenomenon as outstanding as the dissociation between a sense of pain and a sense of touch should have its substrate in the periphery, whereas no such substrate could be found in the manner outlined above in the periphery.



The same holds good if we try to account for a dissociated sensibility behind a severed root on the basis of the *transitional fibres* hypothesis. Then, too, predominantly thick, myelinated (touch-conducting) fibres would remain behind in areas where only tactile sensations are experienced. This seems to us unlikely, for the same reasons. Moreover, this would necessarily imply that transitional fibres serve principally or exclusively for the transmission of tactile stimuli, whereas it was precisely by monoradicular mechanical pain stimuli that the *possible* existence of transitional fibres came to be seen as a *probability*.

Lastly, surely one would expect some sort of arrangement of thin and thick fibres in the peripheral conducting pathways in relation to the transitional fibres? Yet there is a random grouping of thick and thin fibres in the peripheral nerve (Ariens Kappers) and in the root close to the ganglion (as we ourselves have observed), while the lateral arrangement of thin fibres in the root close to the spinal cord (Ranson) is too distant from the periphery and is associated with the junction of these fibres in the lateral pathway in the spinal cord.

Summing up the foregoing, we come to see the matter as follows: The overlapping of root areas is a generally recognised fact, ascribed by Foerster to the through-growth of fibres alien to the segment, whereas we attribute it to the function of (transitional) fibres belonging to the segment. The dissocia-

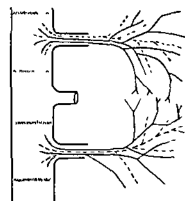
sensitiveness; then the gross sense of touch and finally the delicate sense of touch and of pain. The failure to distinguish between these two different modalities of painful sensation caused confusion at first and is probably in part responsible for the non-correlation of certain experiments.

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Hypalgesia
Thermanaesthesia

How are we to account for this dissociated sensory disturbance? And what significant inferences can we draw from it for practical work, notably in the case of the partly injured or compressed root? It seemed obvious that the explanation would be found in the *overlapping* of the peripheral sensory innervation and that the dissociation could more especially be related to the opinion held by Foerster, *viz.*, that the "dermatome of touch" is larger (or seems larger) than the pain "dermatome" ("dermatome" = root area). For it follows from this that the larger tactile dermatomes of two neighbouring roots may extend sufficiently to overlap the denervated area behind the severed root, whereas the smaller dermatomes of pain are not able to do so. In that case a hypalgesic or analgesic area will be formed between the two intact roots, and that correlates with the findings in practice. The same applies to other modalities of sensation, in the sense that the thermaesthesia dermatomes, for instance, are smaller still.



Pain . . .; Touch ———

Re 2. It is by no means clear how we are to understand the existence (or absence) of dissonance. Theorising, we might start from the following premisses: Dissonance is brought about by the discrepant transmission of one stimulus in two separate fibres, *e.g.*, rapid, short transmission and slow, prolonged transmission. The two different impulses are at odds with each other during integration in the spinal cord and this produces the sensation of pain. If one of these two kinds of fibres is lacking, there is no dissonance and, therefore, no sensation of pain. Understood in this way, the extension of the sense of pain would again be anatomically bound to the distribution of one kind of fibre. As we have seen, this would appear to be inconsistent on anatomical grounds (different fibres *not* found in denervated root area) and for physiological reasons (elasticity of the pain dermatome dependent on the intensity of the stimulus).

Dissonance obtains because one particular kind of fibre is capable of conducting two non-according impulses. (Granit, p. 287 does not consider it beyond the bounds of possibility that a fibre should be able to do this.) We could then call these fibres "pain fibres" and, according to former views, these would be thin, poorly myelinated fibres. But this again entails anatomical fixture of the area of peripheral pain perception and the same counter-arguments come into force.

It is our personal opinion that, while dissonance in the transmission of a stimulus may make it easier to understand how a pain stimulus is conducted, the theory does not account for the dissociated sensory disturbance behind a severed root.

Re 3. We would not have brought these considerations forward if we had not had another suggestion to make which might account for this dissociation between the senses of pain and touch, without doing violence to known physiological facts and practical experience; nor does this suggestion contravene the transitional fibres to which we have referred.

The skin is afferently connected with the spinal cord through the posterior roots; in saying this we disregard *how* a pain stimulus, for instance, is conducted and by which fibres. "The continuous stream of incoming impulses sustains for the greater part the background activity of the spinal cord. Involvement of posterior roots slows down the electrical activity of the spinal cord" (Ten Cate *et al* and others, see Droogleever Fortuyn, p. 573). "The effect of a stimulus results from the stimulus plus the background activity of the elaborating system" (*ibid.*).

From the area behind a severed root the spinal cord receives a stream of impulses of varying strength, depending on the degree of overlapping and

tion between the senses of touch and pain is likewise a fact, but a different one. Neither the classical overlap according to Foerster, nor the transitional fibres as such can account for the latter phenomenon. Regarding this apparent contradiction, let it first be said that generally the pain dermatome only *seems* to be smaller than the tactile one; the dissociation between the senses of touch and pain is mainly quantitative. A strong pain stimulus in a hypalgesic root area is perceived more distinctly than a weak one; an intense pain stimulus in an "analgesic" area previously explored with "physiological" pain stimuli produces a sensation of pain nonetheless. Hence the expanse of pain dermatomes behind a *completely* severed root would depend more or less on the intensity of the pain stimuli applied for testing (presuming the sensorium and/or the psychic condition of the person tested to be constant). This does not make sense, at any rate if the sensation of pain is to be tied to a given anatomical structure. If we call a fibre structure of this kind "the pain fibres", the ineluctable conclusion must be that a pain fibre must be present and must function at the place where a pain stimulus touches home.

If the extension of a pain dermatome is dependent upon the distribution of such a pain fibre system, this would rule out the perception of *any* pain stimuli, even intense ones, outside this system. The elasticity of the notion "pain dermatome" under varying circumstances in practice, among other things as regards the intensity of the stimulus, inclines us to think rather that the extension of the peripheral pain sense is subservient to the *function* of the peripheral nervous system; and this implies, either that there is no separate pain-fibre system, or that the pain-conducting fibre system is ubiquitous, but does not function everywhere – *e.g.*, behind a severed root.

Weddell, Woollard and others described extensive, thin, poorly myelinated "pain fibre plexi" in the skin Nowadays, the view that thin and thick fibres are pain and tactile fibres respectively is no longer strictly adhered to. Without going fully into the arguments, we would say that the perception of various modulations of sensation is ascribed to a difference in the transmission of the stimuli, a sensation of pain, notably, being the result of disharmony in the transmission of stimuli (Weddel, Laruelle and others).

If a pain stimulus is not experienced in a denervated root area, this may be due to·

(1) lack of means of communication with the spinal cord. This would not apply here, because tactile stimuli do get through;

(2) disturbed function, *i.e.*, a pain stimulus does not produce dissonances and, therefore, any sensation of pain

(3) The fact that the pain stimulus remained below the threshold.

in the intact neighbouring root areas will "appear to be larger than the pain dermatome" (Foerster).

This at the same time accounts for the elastic extension of the pain dermatome, depending on the intensity of the pain stimulus, since an intense stimulus requires less background activity to get over the threshold and the area in which there is already sufficient activity will prove to be larger than for a milder stimulus.

On the basis of this hypothesis, which is not tied to anatomical limits, we consider the dissociated sensory disturbance behind a severed root to be an exogenously organic functional disturbance. The root area continues to be connected with the spinal cord by a receiving and transmitting apparatus, with, admittedly, individual quantitative differences, depending on the thickness of the root, the degree of overlapping or the distribution of the caudal fibres through the severed root and its neighbouring roots. Recognition of the fact that an *exogenous*, *organic* lesion of a root changes the *function* of residual intact fibres takes our thoughts back to the miraculously rapid recovery from sensory disturbances after section of a posterior root already referred to (*loc. cit.* p. 320) and observed both by others and the author. This recovery within a few days or weeks cannot be explained on an anatomical basis (no regeneration of post-ganglіar fibres; too short a time for reinnervation by growth of peripheral nerve fibres). A temporary disturbance of *function*, or the coming into *function* of a residual system provides explanations which are compatible with the foregoing hypothesis.

What are the significant inferences we can draw from this for medical practice confronted, more especially, with a *partly injured* or a compressed root?

During neurological examination of patients who, as verified later at operation, were suffering from root compression, we found pre-operatively a variety of disturbed modulations of feeling in this root area. This is a known phenomenon. Zervopoulos (Spiegel 1957, p. 328) has found diminution in vibration sense as a reliable finding for localisation of lumbar disc herniations, by which he was able to differentiate between disturbances of roots L.5 and S.1. In our own experience the sensation of pain was apparently affected first and to the greatest extent, but the results of more accurate examination reveal the following sequence.

1. Sense of vibration.
2. Thermaesthesia.
3. Sense of pain (head-point sense (discrimination between head and point of a pin) still intact)

irrespective of the way this overlapping comes about (through-growing fibres from an alien segment (Foerster) or transitional fibres of the same segment, which explain overlapping in a different way).

There is no reason whatever for supposing that fibres which continue to carry stimuli from a denervated root area do so in any other way than before the section of the root involved. Irrespective of how it is conducted, by what kind of fibres or in relation to what modality of sensation, the stream of incoming impulses in support of the background activity is *quantitatively smaller via* the fewer remaining fibres from a denervated root area than from an intact one. Now, a peripheral stimulus requires an undercurrent of a certain strength to pass over the threshold of the sensory modality involved. *This stimulus threshold does prove to be different for the senses of touch and pain (for example), notably being higher for the sensation of pain.* (Ariëns Kappers, p. 784.)

Modern electro-physiological research workers are reticent on the comparability of sensory perceptions (Walshe). The sensations of pain and touch are said to be just as incomparable as are, for instance, the perception of the colour "red" and the experience of pain. The words "stimulus threshold", indeed, are scarcely used at all in Granit's book. If, instead, we say that the chronaxie for the pain stimulus is long and short for the tactile stimulus (Ariens Kappers, p. 784, Droogleever Fortuyn, p. 573, Klein, p. 36, Granit), we are neglecting the fact that these two concepts are not completely comparable. In this respect, positive data on the rheobase would be more instructive. We have to bear in mind, however, that the point here is not the theoretical interpretation of the stimulus threshold in electro-physiological experiments, but the *practical significance of this concept in daily life and in neurological examination* It is this meaning that led the research workers prior to the period of electronic electro-physiological examination to make statements about "the threshold of pain" (Ariens Kappers and Foerster, p. 364); and the concept is used in the same sense when mapping out the pain dermatome. This can be clarified by the description of a simple test Heat a sharp metal object, such as the point of a pair of compasses, then only just touch the healthy skin with this heated point The only sensation perceived will be one of touch If slightly grea[illegible] experienced to the exclusion of the sensation of [illegible] but if the contact is more prolonged, [illegible] further, a sensation of pain will be produced at that moment and the previous tactile and thermal stimuli will be supplanted in a varying degree. References to a "stimulus threshold" in the past and in practice derive from such experiments. (NB. "Warm" may be so hot that a burning pain is produced eradicating the tactile stimulus, sharp pain stimulus and thermal stimulus Hence the object used should not be over-heated.)

Apparently, the pain stimulus requires greater background activity from a given area than the tactile stimulus to pass its specific threshold. Consequently, the area of neural loss behind a severed root, where the total background activity is too trifling to sweep the stimulus across the threshold, will be larger for the sensation of pain than for that of touch, therefore, the tactile dermatome

There has not been found to be a corresponding arrangement of thick or thin fibres in the transmitting apparatus compared to the fairly consistent chronological order in which disturbance of the sensory qualities occurs. However, if we subscribe to the view (Ariëns Kappers, p. 789, and others) that *thick fibres are more sensitive to pressure* than thin ones, there is no necessity for a particular arrangement of thick and thin fibres. Granit (p. 46), giving a survey of the relevant literature, considers this point and believes that it is better to ascribe greater sensitiveness to pressure to rapidly conducting fibres generally. Now, seeing that a given fibre structure is held to be responsible for the transmission of a certain modality of feeling, then the task of conducting the sense of touch was and is assigned to the thick, or rapidly conducting, fibre. This means to say that *if*, through pressure on a root, any dissociation occurs in the sensory disturbances thus brought about, it is precisely the sense of touch which will be most affected. But this is incompatible with the facts as found in practice, *viz.*, that the sense of pain is disturbed in a greater degree than the sense of touch.

Once again we must point out that it is not only through total section of a posterior root, but also through a partial lesion, *e.g.*, compression, of a root that the stream of incoming impulses is diminished quantitatively, with consequent less background activity of the spinal cord. Within the context of the foregoing, we have then to suppose that the modulations of sense with the highest intrinsic stimulus threshold will no longer be able to pass this threshold; consequently, pain stimuli and thermal stimuli will be less easily perceived. If the background activity is increased – preferably by moving muscles of the same segment – pain stimuli and thermal stimuli will be experienced sooner in a hypalgesic and a thermhypaesthetic area, respectively, provided, of course, sufficient transmission to the spinal cord has remained. This we were once able to prove by a simple test. We fixed a warm metal plate to the thermhypaesthetic skin behind a radicular lesion (which later proved to be caused by a hernia); when the patient was at rest, he did not feel the warmth of it, but on moving he did.

As yet we have no ready explanation for the fact that, as regards the extremity dermatomes, the *distal* parts are disturbed sooner and more severely than the proximal ones and, as far as the rump is concerned, the ventral areas suffer sooner than the dorsal (Biemond, pp. 18 and 236). It is still uncertain whether the distal and ventral parts, respectively, exhibit less overlap, or whether the sensory fibres deriving from them are more vulnerable (see ditto).

On the analogy of the foregoing, one is obviously inclined to think of less background activity from these distal and ventral parts of the segment.

In fact, Klein's *work on sensory chronaxie* (p. 25) affords some support for this view. One of the things he established by rheobase and chronaxie determination was that

4. Most delicate tactile sense (= head-point sense was already lost).
5. Sense of discrimination.
6. Sensitivity to electric current.
7. "Deep" feeling – sense of posture.
8. Gross tactile sense.

The customary means used for routine neurological examination were applied for these tests (tuning-fork, tubes of hot and cold water, pin, cotton-wool, etc.). We tried to maintain an even intensity of stimulus. It at once became apparent that this was not the sequence in all patients; also that individual differences occurred. Thus sometimes the sense of vibration was less disturbed, or in a smaller area, than the sense of pain; occasionally we found the tendency towards disturbance of the sense of discrimination to precede disturbance of the sense of pain in chronological order. Hence, although the above chart provides only a rough indication, it does in the main reflect the observed sensory disturbances after total section of a posterior root, *viz.*,

1. Thermanaesthesia.
2. Hypalgesia.
3. Little or no disturbance of the tactile sense.

The first conclusion to be drawn from this is that, to detect a radicular lesion in practice, the test for sense of pain, or of temperature, is more important than that for sense of touch.

Our next question must be how compression of a root (*e.g.*, by a hernia close to the ganglion) can bring about a sensory disturbance of a dissociated character, for this is truly an amazing phenomenon. For, the entire root is, after all, compressed, either by the hernia or bone margin, or by counter-pressure by the bone situated above the root. Moreover, at this site there is no orderly arrangement of thick and thin fibres, as we know. Let us compare roughly the chronological order in which the disturbance of certain modulations of feeling occurs with known physiological data on the course of those fibres to which the transmission of these modulations is ascribed:

	Diameter of fibre	*Arrangement of fibres in*			
		Nerve root			*Spinal Cord*
			Near ganglion	*Near spinal cord*	
Sense of vibration	Thick	—	—	Middle	Uncrossed
Sense of pain	Thin	—		Lateral	Crossed
Most delicate sense of touch	Thick	—	—	Middle	Uncrossed
Electric stimulus	Thick	—	—	Middle	Crossed?
"Deep" feeling	Thick	—	—	Middle	Uncrossed
Gross sense of touch	Thin	—	—	Lateral	Crossed.
	(Granit)				

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psychogenic sensory disturbances are distinguishable from those of organic origin by a labile stimulus threshold and a varying size of disturbed area He was able to differentiate cerebral and spinal disturbances from disturbances of a peripheral nature by a rising stimulus threshold in the one case and not in the other, due to stimulus fatigue.

Convincing evidence was forthcoming that, in the event of sensory disturbance, virtually all qualities are disturbed in equal measure, or dissociated, *i.e.* when senses of pain and temperature are disturbed, the sense of touch remains intact, or the senses of pain and temperature are disturbed in a greater degree than the sense of touch. Klein points out once more that the chronaxie for sense of touch is lower than for the other modalities of feeling (p. 37, with references).

Of particular interest in this context are the sensory rheobase determinations for different parts of the body with sensibility undisturbed (Klein, p. 27). The highest values for the rheobase were found in the rump and the lower extremities, a fact that was attributed to a difference in physiological irritability (*i.e.*, height of stimulus threshold) of certain areas of the skin. This finding, *viz.*, lower extremities and rump, does not agree with the distal segmental parts and ventral areas of the rump postulate, but it follows from Klein's *observation that there are areas of the skin where the stimulus threshold is* physiologically higher or lower.

Upon simple mechanical stimulation (tickling), the reactions of the subject already betray heightened irritability, *i.e.*, a lower stimulus threshold in the proximal parts of the extremities and the back than in the distal parts, with the exception of a few areas of the skin, such as the tips of the fingers and soles of the feet. We have verified this by determining the sensory rheobase in some persons, finding that the distal segmental parts do in fact appear to have a higher stimulus threshold. Whether one ascribes this to less overlap or to extension of the peripheral dermatomes, the fact remains that a higher stimulus threshold requires a stronger stimulus. Of two theoretically equal stimuli, that at a peripheral part of a segment will have the greater need for the undercurrent of background activity. Therefore, if this undercurrent is cut off or reduced by a root lesion, this will be most clearly noticeable at a distance; put differently. under theoretically equal conditions, stimuli are at first not perceived in the distal parts of the segment, where sensibility is disturbed first and most in degree. It is compatible with this that the dissociation of sensory disturbances due to a lesion of peripheral nerve tissue is manifested first and foremost in the distal parts of the innervation area involved.

Functional derangement (decompensation) of the central nervous system

In the light of what has been said with regard to organic functional disturb-

ances without an anatomical substrate, a certain derangement of the central nervous system can be defined as pseudo-psychogenically organically functional.

We often see causalgia, neuralgia and phantom pain as multi-conditioned phenomena, by which we mean that endogenous as well as exogenous biological and exogenous psychological factors are involved in their genesis. Here we shall only mention these disorders, assuming that the typifying clinical picture is known.

1. *Causalgia*, causalgic sensations, parapathic and hypersensitive sensations which are projected into the innervation area of partly diseased or injured peripheral nerve tissue (see *Biemond*, 1956, pp. 12 and 13).

2. *Neuralgia* is the term used to convey an irritated condition in the innervation area of a peripheral nerve, without a known anatomical substrate. Several kinds of neuralgia, however, are known only to develop after a partial lesion of peripheral nerve tissue (*e.g.*, intercostal neuralgia after herpes zoster or root compression in foramina, lumbo-sacral neuralgia after operation for an inguinal hernia).

3. *Phantom pain* is subjectively projected into a no longer existing part of the body. Haber (see Spiegel, 1957, p. 543) advances arguments in favour of the theory of the central origin of phantom limb. The phenomenon might sometimes be combated by extirpation of a neuroma in the amputated nerve; but it may well prove quite useless, or else the sensations return long before a new neuroma could be formed.

The specific character of the above syndromes is far from being accounted for by clearly recognisable lesions of the nervous system, the results of which are usually manifested in another, known manner. These phenomena cannot be explained by the primary nerve lesion itself; it is presumed, rather, that a change in *function*, say of the spinal cord, is responsible for them.

The three disorders enumerated have the following characteristics in common:

a. They arise only in association with personality structures predisposed psychically to this sort of thing (re 1 and 3: personal view), or the complaints are psychogenically overshadowed (re 2: Biemond, p. 334, re 3: Blood, see Spiegel, 1957, p. 543).

b. Mild stimuli produce disproportionately violent sensations; sometimes, in fact, such sensations arise spontaneously.

c. After a lesion of, or operation upon, the nervous system, there is an interval free from symptoms, varying from individual to individual, but lasting on an average one to two weeks.

d. An undercurrent of afferent stimuli is to be expected from the area behind

the partly injured nerve, or missing part of the body, this undercurrent possibly being increased at first, lowered later and increasing again upon regeneration. Hence the background activity of the spinal cord has changed, or varies.

It takes about the same time (one to two weeks) for a residual function behind a severed posterior root and for residual pathways of the sympathetic system to come into operation.

Taking this into consideration, could the above syndromes be attributable to disturbed adaptation of the central nervous system (in this case the myelum) to the varying background activity? The waxing and waning and build-up again of this activity, as well as replacement by other systems, suggest a temporary or permanent derangement (decompensation) to which sensitive individuals (with a central nervous system somewhat different from the usual?) seem to be predisposed. Put differently, the myelum is embarrassed with the excess or inadequacy of background activity and diverts this – or the overcompensation from spare pathways – to other areas.

We have already mentioned the hyperirritability of the innervation areas adjacent to an injured root which can be observed in practice. Haber (see Spiegel, 1957, p. 543) finds after amputation that stump tactile sensitivity exceeded that of homologous parts of the sound limb in all of three measures: light touch, two point discrimination and point localisation.

The results of a variety of surgical interventions (chordotomy, tractotomy according to Sjöquist, rhizotomy), intended as therapeutic treatment of these syndromes, are dubious, unpredictable and often only transitory. With the above postulate in mind, it is easy to see that interventions of this kind subject the already deranged central nervous system to a fresh shock. The changed, *i.e.*, pathologically adjusted, intrinsic rhythm is temporarily lost. In course of time, the effect of the shock works itself out; the intrinsic rhythm that has to be built up anew may be different (which means to say not less disturbed, but *functioning differently* compared with the pre-operative rhythm), so that possibly the pre-operative symptoms no longer occur. As a rule, however, the central nervous system with its own rhythm will again begin to function with a new deranged rhythm, with the result that – as we find in practice – the symptoms recur after a time.

There is evidence that causalgias and so forth are prone to develop after partial lesions of peripheral nerve tissue containing many sympathetic fibres. It is for this reason that the decompensation of the central nervous system is attacked to best advantage by interventions which effect an integrating change of function in the local sympathetic system.

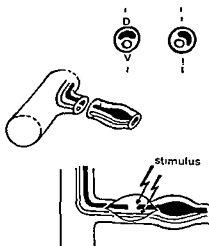
Some facts emerging from the foregoing considerations

1. Psychological and psychiatric insight is required for the appraisal of sensory disturbances.
2. In neurological examination the sense of pain is more important than the sense of touch.
3. A functional disturbance, among other things, may be brought about in the morphologically intact nervous system by an exogenous, organic cause; this may account for the fluctuating picture of sensory disturbances and their rapid recovery.
3a. A lesion of one part of the nervous system may cause a change in function of morphologically intact other parts.
4. Diminution of the background activity due to a partial root lesion (*e.g.*, root compression) can be accepted as the cause of several phenomena, such as dissociated sensory disturbance in the area of the affected root, and earlier and more marked sensory disturbance in distal parts of the segment.
5. Causalgias, neuralgias, phantom pains and allied syndromes are some of the results of decompensation of a central nervous system predisposed to them. A point to be considered is whether the varying degree of background activity has something to do with this.

Motor Disturbances

33–44% associated with hernia

At rhizotomy in the root sheath it is to be seen that motor and sensory fibres in the extradural root, close to the ganglion, still run in separate bundles. Up to that point the ventral position of the motor anterior root fibres with respect to the spinal cord is, largely, maintained. After the dorsal plate of the root sheath has been opened, stimulation shows that the sensory bundle(s) is (are) situated dorsally. Moderate mechanical or electric stimulation of the root distal to the section of this(these) dorsal (sensory) bundle(s) produces muscular reactions, but is no longer experienced as pain.



It is generally assumed that sensory dis-

turbances occur more frequently than motor disturbances. In our material we found, associated with hernia verified at operation:

Reflex changes	44%
Paresis in	34%
Atrophy in	4%
Micturition disturbances in	6%
Sensory disturbances	41%

This difference seems to us too trifling to call for a given mechanism (greater sensitivity of motor or sensory fibres, counter-pressure on sensory fibres by the bony dorsal margin of the foramen) to account for it. Perhaps a contributory factor is the greater impression made by minor sensory disturbances during the neurological examination than barely perceptible diminution of power. Others (Kostić, p. 75, Roeder, p. 8) have it that the reflex pathways are more sensitive, as they are often silent before there is any sensory disturbance.

Reflex changes

(About 44% associated with hernia. About 2–5% in a radicular syndrome without hernia.)

The incidence of *essentially inactive* reflexes, multiple or unilateral, congenital or becoming manifest during lifetime, is approximately 1 in 10,000 (Wertheim Salomonson, Biemond, see Verjaal, p. 1186). It can, therefore, be ignored in practice and in statistics; moreover, if it occurs without a known cause, the possibility is always present of its being the result of a cured disease (old hernia: Reischauer, deficiency disease; beriberi: Van Wulffte Palte). Congenital essential absence of reflexes is also regarded as a manifestation of the status dysrhaphicus (Biemond, Bijl). The phenomenon is in a similar category to a "pseudo-Babinski" due to a Dupré's motor debility (=not fully functioning pyramidal tract).

To our way of thinking, phenomena of this kind are to be classed with the manifestations of the psychosomatic entity to which the degenerative back also belongs, there is no available evidence to support this supposition.

In a radicular syndrome, abnormal tendon reflexes may be accompanied by a so-called *pseudo-Babinski* We have then to consider:

1. Dupre's "débilité motrice"
2. Signe d'éventaille (supranuclear lesion acquired in youth)
3. Paradoxical ankle r. (= peripheral Babinski) through a peripheral or nuclear lesion (S.1, S.2, S.3) of the afferent extensors.
4. Abnormality in the foot (bone or muscles).

5. Hyper reflex action through over-stimulation (vanishes after herniatomy, for instance) (abdominal and cremaster reflexes are normally positive) (Foerster, p. 147).
6. Vascular disturbances of the cord due to obliteration of an arteria radicularis lumbalis (De Sèze *et al.* 1957, p. 1788).
7. Suprasegmental reflexes above a peripheral lesion??

A reflex may be lacking without an associated paresis, atrophy or any electro-physiologically demonstrable change in the irritability of the muscles. Reflex action is dependent upon the co-ordination of various elements of the nervous system. The fewer the functioning efferent fibres, the stronger will the afferent stimulus have to be. When the inhibiting influence of the supranuclear system has been eliminated, a hitherto negative reflex can become positive again. "Every element of the nervous system acts as a check upon its neighbour; if one system falls into abeyance, another enters into a condition of heightened irritability, with greater (reflex) liveliness as the result". With these words, which we have not quoted verbatim, Foerster (p. 148) implies that there is a relatively functional interdependence of the systems in the nervous system. These we are to regard as the primary cause of variations and the changing intensity of the reflexes. A reflex tract may be so severely injured that the reflex is definitely or absolutely negative. Conditions, however, may produce a varying degree of irritability of the afferent system, of the sensorium as a whole, or through the psyche of the patient in his entirety as a single unit, so much so that the function of the reflex tract is enlivened and, let us say, a somewhat diminished reflex appears to be normal again. Jendrassik's handclasp shows that reflex activity is liable to vary under the influence of psychological factors.

We consider *hyper reflex action* in root areas attacked by cooling (Zukschwerdt, p. 175) to be an organic functional condition. All-round heightened reflex activity may be pseudo-psychogenically organically functional, but seems mostly to be pseudorganically psychically functional (as, for instance, in the constitutionally highly strung)

We noted hyper reflex action as the result of over-excitability of a neighbouring root, which at the same time was manifested by hypersensitivity all round.

The reflex pathways are thought to be:

	Ankle reflex			*Knee reflex*		
Armstrong		S.1	S.2		L.3	L.4
Bradford and Spurling	(L.5)	S 1	S 2	(L.2)	L 3	L 4
Déjerine	L 5	S 1	S 2	L.2	L.3	L.4
Hoelen		S 1	S.2	L.2	L.3	L.4

	Ankle reflex					Knee reflex					
Krayenbuhl			S.1	S 2			L 3	L.4			
Lewin		L.5	S.1	S.2		L.2	L.3	L.4			
Roeder			S.1	S.2			L.3	L.4			
Reischauer			S.1	S.2			L.3	L.4			
Tilney and Riley		L.5	S.1	S.2		L.2	L.3	L.4			
Wechsler			S 1	S.2		L.2	L.3	L.4			
Foerster											
(p. 142) afferent	L.4	L.5	S 1	S.2				L.4	L.5	S.1	S.2
efferent			S.1	S.2	S.3	L.3	L.2	L.4 and other roots.			

The opinion has been gaining ground in recent years that a diminished or negative ankle reflex is associated with a lesion of the S.1 root and points to a hernia at L.V – S.I; a diminished or negative knee jerk is thought to be due to a lesion of roots L.3 and L.4. Some authors almost fully subscribe to this view; *e.g.*, Armstrong, p. 93: "At the L.IV – L.V and L.V – S.I disc level, the knee jerk is not affected". Reischauer, p. 35: "Die Wurzel L.5 ist reflex-ledig. Störung (des Achilles Reflexes) muss unbedingt auf die Lumbosacralscheibe bezogen werden. Gestörter PSR = L.3/4, keine Reflexstörung = L.4/5, Achilles Reflexstorung = L/S". Krayenbuhl (p. 37) is another who ties up the knee jerk with roots L.3 and L.4; a hernia at L.IV – L.V is supposed to diminish the ankle jerk, but not the knee reflex. Bradford and Spurling, p. 81, and Roeder, p. 8 are of the same opinion.

Although few express themselves so unconditionally, many a neurologist and neurosurgeon takes reflex changes into account when trying to ascertain the level of a lesion.

There is information to hand in the literature, on the basis of which this postulate can be reduced to its proper proportions. We shall list some of these roughly, together with our own data.

Associated with verified hernia we found reflex changes in	44 %
Bradford and Spurling (p. 79)	50 %
Kostic (p. 77) roughly	40 %
Krayenbuhl (p 36)	43 %

In our material, reflex changes (6 %) were also observed in patients who, suffering from a radicular syndrome, were found at operation to have no hernia nor demonstrable lesion of the roots, the so-called "negative explorations". This is in agreement with Ståhl's findings (2–5 %). Hence, in the proportion of 6 to 44, the patient might show reflex changes which were not caused by a hernia or root compression. In a word reflex changes in association with a radicular syndrome are not pathognomonic for a hernia. This conclusion would seem to conflict with what Voris says (p. 122), *viz.*, "In the

cases in which I failed to find a disk protrusion, such objective neurologic findings (absence or impairment of the ankle jerk) were never present".

The ankle jerk seems to be affected more often than the knee jerk:

	Ankle<	*Knee<*	*Ankle < and Knee<*
Bradford & Spurling (p.79)	>50%	<10% (?)	
Kostić (p. 77)	39%	41%	
Ursula Clinic	55%	28%	32%

These figures should be compared with the following (Waris, p. 44).

	Knee jerk – or <
Malmrose	9%
Wiberg	11%
Waris	7%

Reflex changes occurred in 44% of our material; dividing these (in accordance with the findings at operation) into purely S.1 and L.5 lesions, we get the following figures:

		Ankle – or <	*Knee – or <*	*Ankle – or < and Knee – or <*
Ursula Clinic:	S.1 lesion	36%	10%	10%
	L.5 lesion	19%	18%	22%
	Hernia L.III – L.IV		32%	

If arranged as follows, these findings can be compared with those of other authors:

		Ankle – or<	*Knee – or <*	*Ankle normal*
	Ursula Clinic	36%	10%	
	Kostić	26%	22%	
S.1 lesion	Bradford & Spurling	80%		
or	Ståhl (p. 144)	56%	0%	
Hernia L.V – S.I	Friberg			18%
or	Krayenbuhl			20%
S.1 syndrome	McCraig & Walsh			32%
	Burns & Young			39%
		Ankle–or<	*Knee–or<*	*Knee normal*
	Ursula Clinic	19%	18%	
	Kostić	10%	18%	
L 5 lesion	Bradford & Spurling	25%		
or	Ståhl (p 144)	16%	34%	80%
L 5 syndrome	Burns & Young	33%		
or	Krayenbuhl	33%		
	Friberg	55%		
Hernia L IV – L V	McCraig & Walsh	63%		
Hernia L.III – L.IV	McCraig & Walsh		49%	
	Ursula Clinic		32%	

		S.1 syndrome or Hernia	*L.5 syndrome or Hernia*	*At operation*	
				No hernia	*Hernia L.III – L.IV*
		L.V – S.I	*L.IV – L.V*		
Ank. – or<	Ståhl, p. 144	62%	16%	5%	
	Ursula Clinic	36%	19%		
Kn. – or<	Ståhl (7 cases)		55.5%	28.2%	14.3%
	Ursula Clinic	10%	18%		

Although the data in the above charts agree to some extent, they are mutually contradictory in some respects, especially in relation to the knee tendon reflex. As far as we were able to ascertain, these data were collected with *reference to lesions of the root or disc in question verified at operation.* We have no acceptable explanation to offer for the fact that a considerably larger or smaller number of knee jerk impairments were found in the above series.

The knee jerk impairments recorded by us were checked up in the St. Ursula Clinic and the University Clinic at Nijmegen in the course of the years independently of each other by eight experienced neurologists; hence there can be no question of a one-sided interpretation of the reflexes by, say, one examiner.

The figures recorded by Kostić (Belgrade, Jugoslavia) and those presented by the Ursula Clinic show a striking resemblance; both sets refer to cases verified at operation; also, both series relate to patients from whom, in the course of differential diagnosis, essential sciatica, lumbago, rheumatism and fibrositis had been eliminated and who, after having undergone conservative treatment, were, on being admitted, more or less differentiated as being suspected of having a disc hernia.

It is our personal opinion that a negative or impaired *knee jerk* points to a lesion of motor and/or sensory fibres of the (L.3), L.4, L.5 or (S.1) root, or else of several roots at the same time. *Hence a changed knee tendon reflex has no significance in regard to the level of a lesion*, and we do not except the L.5 root. After the following surgical interventions on patients whose knee jerks were normally positive pre-operatively, this reflex was impaired or negative:

5 times temporarily impaired after foraminotomy for root L.5.
3 times temporarily impaired after stripping L 5 from adhesions
1 time negative, impaired after stripping L 5 from adhesions.
3 times impaired, 21 times undisturbed after selective sensory rhizotomy of L.5.
Once negative, once undisturbed after total rhizotomy of L.5.

According to Foerster, the intensity of a reflex is determined, for one thing, by the sum total of afferent stimuli. It is therefore worthwhile considering whether the diminution of the knee jerks may not be due to reduced afferent function of more than one root (*e.g.*, L.5 and S.1), or of one root if this covers an exceptionally large root area. We should at the same time consider, also with regard to the efferent reflex tract, whether the many facts concerning the cauda equina which have come to light since sciatica has been treated by surgery, suggest that the reflex tract of the knee jerk is *not* confined to roots (L.2), L.3 and L.4, as in the current view.

We find that an impaired or negative *Achilles tendo reflex* is by no means only related to the condition of the S.1 root; a diminished or negative ankle jerk has been noted in association with lesions both of root L.5 and of root S.2. This latter is equally important, because a localised radicular syndrome of the S.2 root likewise occurs. With the loss of root L.4, sufficient stimuli are found to reach the spinal cord to keep the ankle jerk lively if L.5, S.1 and S.2 roots are intact. There is nothing against concluding, with Ståhl (p. 148), that: "A complete absence of the Achilles tendon reflex seems to suggest that the chances of finding the herniation lumbo-sacrally are almost twice as great as of finding it protruding from the fourth disc". *In an individual case, however, we do not attach any definite localising value to a negative Achilles tendon reflex.*

In an elaborately documented study, Ståhl shows that neither a combination of reflex changes with other disturbances, nor interpretation by the first neurological symptom to occur, or to be most violently disturbed later, has any exact localising significance. We shall give a single example. (The criterion for the acceptance of a single lesion given (p. 147) is that a hernia was not detectable at other levels, while the pre-operative symptoms cleared up after excision of the one hernia found. We consider this criterion to be practically acceptable.)

Ståhl (pp. 145 and 147).

	Ach r. – or <	*Paresis of Musc. extensor hall. longus*	*Ach r. – or < and paresis of M. ext. hall. longus.*	
Hernia L.V – S.I	80 %	14 %	35 cases =	50 %
Hernia L IV – L V	15 %	83 %	29	41 %
Hernia L.V – S.I and L IV – L V			4	6 %
No hernia	5 %	3 %	2	3 %
	129 cases	64 cases	70 cases	

		S.1 syndrome or Hernia	*L.5 syndrome or Hernia*	*At operation*	
				No hernia	*Hernia L.III – L.IV*
		L.V – S.I	*L.IV – L.V*		
Ank. – or<	Ståhl, p. 144	62%	16%	5%	
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We can find little to go upon with regard to the variability of motor innervation, more particularly of the Achilles and patellar reflexes, in the relevant literature. Reischauer (p. 35) speaks of "Inkonstanz des Reflexverhaltens". On the whole, the Anglo-Saxon literature does not deal with the consequences of observed reflex differences in a positive way (see, for instance, Bradford and Spurling). Biemond (in a paper read to the Netherlands Society of Neurosurgeons, Nov. 1957), discussing paralyses and pseudoparalyses, estimated the number of motor variations to be 20 per cent. Bradford and Spurling discuss the common neurological misapprehensions (p. 112). The meagre number of patients with positive findings in some groups suggests, they think, that much depends on how carefully the neurological examination has been performed. They draw attention to several contradictions in the literature, including the function of the L.5 root. According to Williams and Yglesias, an involvement of the fifth lumbar nerve alone might result in an extensive weakness of the leg muscles, a statement which Bradford and Spurling dispute on several grounds.

Our reasons for supposing that the motor cauda fibres may be subject to anomalies can be summarised as follows:

1. It cannot be convincingly denied that a non-oedematous, non-inflamed root of unusual girth might also contain more motor fibres than a thin root.

2. We collected records of 13 monoradicular *total* sections:

3 times pronounced paresis 6 times mild (transient) paresis 4 times no paresis	10 times reflex diminution. 3 times reflexes unchanged.

How could this be possible if the number of motor fibres, or their function, in a root did not vary and if an unusually large number of motor fibres did not occur in certain roots?

3. We were able to establish beyond doubt in at least five cases that motor action and the reflexes were undisturbed, in spite of the fact that a root was found at operation which, tested by mechanical and electric stimulation, no longer exhibited motor reaction. (Twice the root lay in granulation tissue after a previous laminectomy and three times the root was atrophic through bone margin or "healed" hernia.) Moreover, this same evidence was presented with striking clarity in a case where, a year previously, root S.1 was completely severed during a laminectomy, as we were able to verify at the second laminectomy. Regardless of how reinnervation takes place in the periphery (see p. 399), along which tracts and from which roots, one may ask, did motor impulses reach the periphery in the cases mentioned under 2 and 3, and what is the cause of the individual differences?

Additional details recorded:

7 cases: Ach.r. < and Kn.r. <
6 times: Hernia L.V – S.I!
once : No hernia.
One case: Kn.r. < and paresis of the M. ext h. longus.
Hernia L.V – S.I!

"Theoretically, for *one* herniation to be able to disturb either both the Achilles and patellar reflexes, or both a reflex and muscle power, it is necessary that either the different functions are controlled by one root, or that more than one root is affected by one herniation. The first alternative is the less acceptable" (p. 146).

This comment suggests the following question: Are there variations in the distribution of the motor fibres among the cauda equina roots of such magnitude as to produce reflex anomalies and unusual pareses? Or, to put it differently, *are there such things as motor transitional fibres as well?* We shall not attempt to advance as many arguments in favour of this idea as we considered necessary to put the case for sensory transitional fibres; nor, indeed, should we be able to, because the conditions are different and the available arguments in favour of motor fibres are fewer. But a few remarks will not be amiss.

1. The sensory fibres grow from the intervertebral ganglia in two directions, whereas the motor fibres do so in one direction only, *viz.*, from the anterior horn to the periphery. If the formation of transitional fibres is influenced by deviations in their course from spinal ganglia and cord to skin and muscles, respectively, motor fibres would seem to have fewer opportunities for deviating in this way.

2. The surgeon engaged on a rhizotomy is at pains to save the motor fibres. As he usually succeeds in doing so, examination after rhizotomy furnishes little information respecting motoricity and the reflexes.

3. Monoradicular irritation of roots during operations did not produce reliable data on motoricity.

4. On the discovery of an impaired reflex not fitting into the rest of the neurological picture, the possibility should always be borne in mind of a residual symptom of a "healed" lesion at another level. Reischauer makes a point of emphasizing this.

5. The subjectivity of the patient (and, be it said, of the examiner) tends to obscure sensibility tests and sensory overlap also plays an important part in these. The fact that the muscles are usually innervated multiradicularly makes it even more difficult to determine a motor root area.

6. Fewer reliable data are known respecting the radicular supply of muscles; that is to say, the myotomes are less well known and their determination is almost impracticable.

Now it appears that Schliack also observed variations, *viz.*,

> Prolonged chronaxie of M. fib. brevis (S.1) in a few L.5 syndromes;
> Prolonged chronaxie of M. ext. hall. l. (L.5) in a few S.1 syndromes;
> Prolonged chronaxie only of M. fib. brevis (S.1) in one L.5 syndrome.

Schliack speaks of L.5 or S.1 syndromes, as the case may be, going by the neurological picture without operative or myelographic verification, *viz.*,

L.5 sensory disturbance in classical L.5 area
radiation of pain in classical L.5 area
paresis and hypotonicity of big toe.

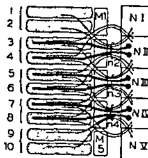
S.1 sensory disturbance in classical S.1 area
radiation of pain in classical S.1 area
Ach. refl. negative or diminished.

It is to be regretted that Schliack's investigation lacks anatomical verification, though it is immaterial to our purpose, because the variations recorded by the writer offer evidence of an antithesis between the neurological syndrome and certain components of the motor innervation Moreover, we have corroborated the basic facts of the investigation by cases which *were* verified at operation. Then, the chronaxie investigation conducted by Griessmann (see Schliack) is known; he also found prolonged chronaxie in association with L.5 lesions verified at operation. (Griessmann determined the chronaxie of the M. tibialis anterior, but not that of the muscles chosen by Schliack for his study.)

In our opinion, the explanation for these variations is an individual variation or anomaly of the motor innervation of the muscles.

Important inferences to be drawn from the chronaxie investigation as to indications for an explorative intervention in the cauda equina roots will be discussed in the chapter on therapy (*loc. cit.* p. 611).

8. The detection of variations in motor innervation raises the question whether these are to be regarded as due to so-called "motor transitional fibres". Apart from the reflex variations and paresis, unusual both quantitatively and in its location, after rhizotomy or other root lesions, we have no experimental evidence. We may, however, point out that Lopes Cardozo's work on the distribution of the filamenta radicularia was also concerned with the *ventral* spinal roots and the myotome. ". . Every (ventral) spinal rootlet also receives fibres from cells which do not correspond with the level of its exit from the spinal cord". "The most cranial part of the myotome (or dermatome) is exclusively innervated by the cranial filamenta radicularia, the most caudal part only by the caudal ones" (pp. 57 and 56). The conclusions drawn



4. As was stated in the foregoing, a lesion of root L.5 sometimes does but usually does not cause a diminution of the patellar reflex.

5. Von Muralt described the neurological picture of 19 patients with transitional vertebrae; the following are one or two briefly summarised facts:

19 "sacralisations"	*Radiation of pain*	*Ach.r. – or <*	*Kn r. – or <*	*Paresis M. ext. h. l.*
+ hernia at L.IV – L.V	7 × heel, lat. edge of foot little toe	6 times		
+ hernia at L.V – S.I	2 × instep		Once	4 ×

From this we see that, in 19 cases of transitional vertebrae, neurological symptoms presented 14 times contrary to the classical views, in five of which motor activity was affected (leaving the diminished Achilles reflex associated with hernia at L.IV – L.V six times out of account). We may provisionally conclude that variations in the motor innervation areas occur more often in association with transitional spinal columns than with normal spines, in the same way as do sensory transitional fibres.

6. We have already referred to reflex varieties (patellar reflex diminution associated with lesion of L.5, but also of S.1), leaving it an open question whether these are to be regarded as genuine anomalies, or whether greater spread is to be assigned to the reflex tract of the patellar reflex (also running over roots L.5 and S.1) than it is usual to do.

7. During our quest for exact indications, we had the good fortune to come upon them in the data relating to a chronaxie investigation carried out by Schliack (p. 471). Several details were correlated with corresponding findings in the St. Ursula Clinic in patients whose malady was verified later at operation.

The chronaxie of a denervated muscle is prolonged; this prolongation takes place 8 to 10 days after a lesion of the peripheral motor neuron from anterior horn cell to muscle. As the majority of muscles are supplied by two or three roots, at first sight chronaxie would not seem to be revelatory with respect to root lesions. Some muscles, however, proved to be supplied monoradicularly – and that almost exclusively:

Musc. extensor hallucis longus, L.5
Musc. fibularis brevis, S.1

In the event of a L.5 or S 1 syndrome, the chronaxie of the appropriate muscle is prolonged

In association with a L.5 *plus* S.1 syndrome, the chronaxie of a group of muscles supplied by both roots is prolonged.

The chronaxie of muscles innervated by L 5 and S.1 is not prolonged if *one* of the roots drops out.

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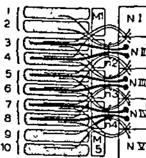
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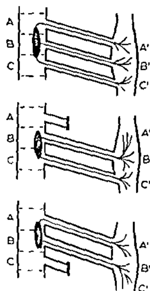
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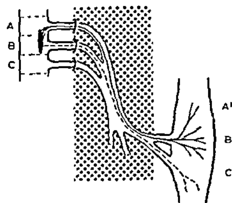
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from his investigation link up with Rabl's view expressed in 1910 (see Corning, p. 532). Every motor spinal root is composed of fibres from three segments of the spinal cord. The fibres connect up temporarily with an adjacent root and in the periphery turn away towards the myotome of its own segment. Corning (p. 533) believes that this also applies to man. To account for the fact that a peripheral nerve contains fibres from two or more segments, it has been suggested that various parts of a myotome shift and merge; in other words, that one muscle comprises parts of several myotomes. Motor innervation from their own segment (*via* transitional fibres) in accordance with Rabl would, Corning continues, make an artificial explanation of this kind superfluous. Certainly, a diagram constructed on the basis of this hypothesis and of our conception of motor transitional fibres would go far towards explaining many of the phenomena brought forward in the foregoing. A variety of *motor transitional fibres* would then easily account for anomalies of quality as well as of location.

The inclusion of the plexus and the peripheral nerve in such a diagram could, certainly, be demanded. It should not be forgotten, however, that the present object is an enquiry into radicular symptoms. Now, a "radicular





symptom" means a symptom in a given part of the periphery caused by the function or loss of function of a root! Like roots a, b and c, the areas A^1, B^1 and C^1 are concrete data. Suppose that a symptom is observed in A^1 and the causal lesion of b is known, then, by definition, there is a relation between b

and A¹. That means to say that, whatever the course of the fibres peripheral to the foramen may be (plexus and peripheral nerve), the functional connection between b and A¹ is an established fact. Provided the conditions be met (symptom in A¹, lesion of b) and the symptom be a phenomenon known to be initiated through transmission or interruption of a stimulus, it is reasonable to accept a connection *via* stimulus-conducting fibres between b and A¹. Therefore, the course of the fibres in the plexus and peripheral nerve is of secondary importance and in this case of no importance. The shaded part on the figure can therefore well be omitted altogether.

Muscle weakness

(This was found to be associated with hernia, in the St. Ursula Clinic in 34%, by Krayenbühl in 41% of the cases.) Muscle weakness may vary from complete paralysis of several groups of muscles to very mild muscle weakness. Wartenberg (see Ståhl, p. 143) includes the quicker tiring of what was at first a normally strong group of muscles. Its detection depends upon the subjective observation of the patient and of the examiner and is rendered more difficult by the occurrence of *pseudopareses*. By these we mean the conscious or unconscious reactions of the patient to painful movements, by which the latter are checked. As a rule, the disturbance appears as a general weakness of the whole extremity, not being limited to any particular group of muscles (thigh, shank, extensors or flexors). This is the initial stage. Later on, when examinations have been repeated several times, the patients soon learn to know which movements are painful and what part of the motor action is expected to have declined. This does not imply that the patient need always be suspected of simulation or of being psychogenically disturbed. It is a different matter, however, if the patient is able to stand on his toes, yet, when supine, declares upon slight pressure to be unable to push away the examiner's hand. Pseudoparesis, psychogenic in nature, should also be suspected if, in attempting to push away the examiner's hand, the patient makes an effort of greatly varying intensity and suddenly gives up the attempt with a certain amount of fuss.

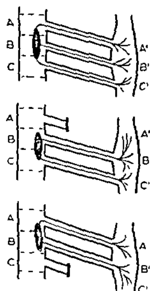
True pareses, however, may also be masked by the execution of jerky movements by groups of remote muscles, by weakening of the antagonists and by the transference of functions to other muscle groups.

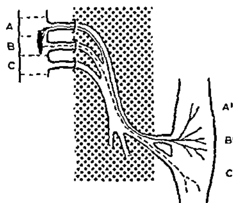
A *partial loss of motor action* may be the result of a partial lesion of the motor root or peripheral nerve. Further, as far as the latter is concerned, it will be necessary to reckon with the variable division of side branches and with the greater vulnerability of certain muscles and nerves (*e.g.*, the peroneal nerves).

from his investigation link up with Rabl's view expressed in 1910 (see Corning, p. 532). Every motor spinal root is composed of fibres from three segments of the spinal cord. The fibres connect up temporarily with an adjacent root and in the periphery turn away towards the myotome of its own segment. Corning (p. 533) believes that this also applies to man. To account for the fact that a peripheral nerve contains fibres from two or more segments, it has been suggested that various parts of a myotome shift and merge; in other words, that one muscle comprises parts of several myotomes. Motor innervation from their own segment (*via* transitional fibres) in accordance with Rabl would, Corning continues, make an artificial explanation of this kind superfluous.

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(see von Muralt, p. 118), who, for instance, attach no localising value to pareses of the extensors. Von Muralt found paresis of the big toe extensors in association with transitional vertebrae four times as the result of a hernia at L.V – S.I.

If we attach an exact value to motor loss symptoms when determining the level of radicular lesions, it should be put, we think, something like this:

Difficulty in walking on the toes: the chances are that the clinical function of the S.1 root has been lost.

Hypotonicity or paresis of M. ext. hall. longus provides some indication of diminished clinical function of root L.5.

We are particularly anxious to repudiate the suggestion that a disturbed peroneal function definitely points to herniation of the L.III – L.IV disc. As against instances given in the literature of a disturbed peroneal function in association with herniation at L.III – L.IV, we could set another series in which a hernia was found at L.IV – L.V, or in which a lesion of L.5 was verified anatomically; the peroneus, which was weak before the operation, gained strength after root L.5 had been freed.

Atrophy of the muscles

(Muscular atrophy associated with hernia was found at 4%)

The influence of a dominating hemisphere should be taken into account when measuring atrophy of the muscles in the *legs*, even though, in the case of a right-handed person, this influence will be less telling as far as the development of the muscles of the (right) *arm* is concerned. We regarded a reduction in girth by 1½ cm of the largest parts of the upper and lower leg in the non-dominating half of the body as being abnormal. On the other hand, we adhered to the principle that a reduction of 1 cm in the girth of the "dominant" lower extremity is abnormal We have, in fact, found upon enquiry and examination that right-handed people do use their right legs more freqently and more actively; this is strikingly apparent in sport, especially in footballers. We know of no instances of right-handed people in whom the left leg dominates.

We do not think the atrophies help us much in the aetiology or locating of a radicular lesion. All-round atrophy of the M. glutaei may point to a given habit (sedentary life); asthenia is accompanied by hypotonicity of the buttock muscles (pendulous buttocks); general atrophy of these muscles is also seen in the elderly (trousers hang loosely around the legs). It might be interesting to seek a connection between general muscular atrophy, general "rheumatic" complaints, increasing stiffness, less inclination to move, disturbances

The muscles of the lower extremities are undoubtedly supplied multiradicularly (Bradford and Spurling, p. 107), with the exception, perhaps, of the M. extensor hallucis longus (L.5) and M. fibularis brevis (S.1) (Schliack, p. 471). Nevertheless, the innervation of some groups of muscles is ascribed so predominantly to a particular root that some authors attach a localising value to their paresis. We quote Bradford and Spurling (p. 109): "The 5th lumbar nerve (injured by 4th lumbar herniations) is the lowest important nerve supplying the extensor (dorsiflexor) muscles of the toes and foot and the 1st sacral nerve (injured by lumbo-sacral herniations) is the highest important nerve supplying the flexor (plantarflexor) muscles, notably the gastrocnemius and soleus".

Kostić (p. 77):	Changes in motility.	
	H.N.P. L.III – L.IV	0.60%
	H.N.P. L.IV – L.V	28.40%
	H.N.P. L.V – S.I	20.0 %
	No disc prolapse	4.20%

(p. 75): "In the case of irritation of the 5th root we usually have paresis of the anterior group of muscles below the knee [while the patellar reflex is usually non-existent or much weakened]. In the case when the first sacral nerve is compressed [*i.e.* when there is protrusion of the disc between the fifth lumbar and first sacral vertebra – the Achilles reflex is more often non-existent and] there is a weakening of the posterior group of muscles below the knee."

Reischauer (p. 35).

Symptom	Root		Hernia
Difficulty in walking tip-toe	S.1, S 2	→	hernia L.V – S.I
Shuffling gait	L.5	→	hernia L.IV – L.V
Difficulty in walking on the heels	L.5	→	hernia L.IV – L.V
Reduced extensibility of big toe	L.5	→	hernia L.IV – L.V
Disturbed peroneal function	L.4	→	hernia L.III – L.IV
Paresis M. quadriceps	higher lumbar roots		
Paresis M. gluteus	cauda syndrome		

Roeder (p. 8).

Lesion		
Lesion S 1	→	Difficulty in walking on the toes
Lesion L 5	→	Difficulty in walking on the heels, paresis of big toe
Lesion L 4	→	Disturbed peroneal function.

However, Roeder believes that monoradicular lesions are barely demonstrable; in his opinion motor disturbances are initiated by lesion of at least two neighbouring roots.

We have not been able to discover constant correlations of this kind in our patient material. In view of the subjective element in the examination and other imponderables, we incline more to the views held by Hiller and Krayenbuhl

of root S.2 also occur (detached hernia from the L.V – S.I disc, hernia from a rudimentary S.I – S.II disc, transitional fibres S.2 to S.1, bony spur in the intervertebral canal for root S.2). In our practice we bear this in mind and, after noticing fasciculations in the calf, inspect the intervertebral canal for the S.2 root to see whether it is clear. In all probability, this precaution has increased the favourable results of surgery for radicular syndromes by 1 % in our statistics for the past two years. Let it be said in parentheses that, from the point of view of the patient, you have to be the one in a hundred cases to appreciate this precaution to its full extent.

According to Reischauer (p. 36), another form of motor hyper-irritability of an injured root is manifested in 52 % by a "spastic tendency" in muscles at rest; tonic spasms, especially in the calf muscles, are induced for minutes at a time upon stretching in bed, during the first movements when rising, or while putting on shoes.

We have found, as have others, that touch and pressure sometimes initiate a painful spasticity of the calf muscles.

Micturition disturbances

(Such disturbances were found associated with hernia, pre-operatively in 4 %, according to Krayenbühl in 4 %.)

Neurogenic affections of the bladder will certainly be due to a combination of factors. Loss of sensory tone and a disturbance in the autonomous and peripheral motor innervation are both involved.

Bladder disturbances may be associated with disorganised function of the anal sphincter and with change or loss of sexual functions (Krayenbuhl, p. 37) The disturbances may be manifested by complete retention or incontinence. If one makes a habit of questioning patients specially on this point, it will be found that minor disturbances are by no means rare. It is certainly necessary to differentiate between these and disturbances of a different nature (*e.g.*, noctural micturition, prolapsus vaginae or other gynaecological symptoms). Even then, the percentage of bladder trouble conjoined with the hernia syndrome is far higher, *viz.*, 19 %, including the loss of a few drops of urine upon any sudden movement, sudden laughing or sneezing, and stress incontinence.

We shall merely point out that lesions of (lumbo)sacral cauda roots, however caused, may lead to these disturbances. If a hernia is the aetiological factor, it is usually medial in the L.V – S.I disc. An abrupt protrusion – during delivery or as the result of a chiropractical manipulation – is notorious in this respect.

in micturition resembling prostate symptoms, declining power, on the one hand and, on the other, a diffuse spondylarthrotic narrowing of the lateral recess in the spinal canal right in front of the intervertebral foramina.

Finally, extensive muscle weakness and atrophy are known in conjunction with hernias in the higher lumbar region (L.I – L.II, L.II – L.III). These are usually bilateral in the thigh, being more pronounced either in the left or right leg. Neuralgia paraesthetica in the clinical history, chronic patellar weakness through atrophy of the M. quadriceps, as well as abduction weakness of the hips, call for extensive neurological and suboccipital myelographic examination, if only for the purpose of differentiating from other cauda-(conus) lesions.

Fascicular contractions

(These were found in association with hernia, in about 1% (?).)

They are rare and it is almost impossible to determine their incidence statistically. They might be observed in association with nuclear anterior horn lesions, seldom with lesions of the motor anterior root and scarcely, if ever, with a disease of the peripheral nerve.

The occurrence of fasciculations is said to indicate hyper-irritability of muscles and nerves, owing to which bundles of muscles contract spontaneously. The contractions can be seen with the naked eye if they occur in muscles immediately under the skin (platysma, M. soleus). The phenomenon is important, not only as an aid to differentiation from a central (pre-gangliar) and peripheral disturbance, but also because fasciculations in the leg muscles are always indicative of a pathological condition, whereas they are not if they occur in the muscles of the rump or the face.

We know of six cases of *clear* fasciculations, *viz.*,

Once behind the thigh and calf (operatively verified hernia)	L.IV – L.V and L V – S.I.
Three times in the calf (operatively verified hernia)	L.V – S.I, lesion S.1 and S 2.
Once in the (calf operatively located lesion)	S 2
Once midline patella and calf (operatively located lesion)	S.2.

This chart shows that fasciculations are prone to occur in the calf muscles. The, probably, greater incidence of fasciculations in association with a lesion of S.2 is perhaps due to the fact that they occur more readily in the thin, spread-out M. soleus (L.5, S 1, S.2) situated immediately under the skin, or are easier to detect there. Meanwhile, we have ascertained that localised lesions

The following example is instructive. At a previous intervention, the indication for intradural rhizotomy (for the relief of radicular pain) had apparently been interpreted somewhat liberally. The patient had retained chronic incontinence ad urinam et ad faecem. At re-exploration, we found that the sensory part of root L.5, the whole of root S.1 and probably a part of root S.2 had been severed on the left, and the sensory part of S.1 on the right. The roots were dissected out of their arachnoid adhesions. Subsequently the incontinence was transformed into retention!

Recovery from neurogenic bladder trouble, which is associated with extensive neurological loss symptoms, is related to the return of sensation. One can say that about three months after recovery of the *function* of the retrogangliar sensory fibres (disappearance of the "saddle" anaesthesia), the autonomous function of the bladder *begins* to return; consequently, depending on the severity of the lesion of the motor fibres, the voluntary functioning of the bladder may be fully recovered in the subsequent three to six months.

Changes to sexual functions

We have mentioned changes to sexual functions in passing. Although these, too, are not rare, they are often not revealed in the anamnesis for understandable reasons. Male patients are prone to offer a ready explanation for the decline in this function.

By "change" we do not only mean a quasi-physiological decline in libido and potency or less intense sensual experience. What we wish to emphasize is that the libido may be given a different orientation, both qualitatively and quantitatively, by a lesion of cauda equina roots, particularly S.2.

Irrespective of whether the condition before or after the intervention is to be regarded as the normal one for the total personality structure of the patient, we noted changes in the sexual behaviour of the patient after manipulations upon the higher sacral roots. A few selected examples will suffice to illustrate what we mean.

Man. Pre-senile, low back pain and radicular symptoms as the result of spondylarthrosis with hernia formation. Overhaul operation. The experience of his hypersexuality was "all he still lived for" and it was for fear of losing it that the patient deferred the treatment as long as possible. Two months after the operation the patient had had no sexual intercourse, although he was free from symptoms and was in good condition What is more, this abstention was not felt to be a disability and the patient did not resent the complete change in his sexual life.

Woman Of middle age, involutional osteoporosis, degenerated disc with hernia formation at L.V – S I; overhaul operation. Insatiable libido, which led to social conflicts Patient's appearance did not facilitate the satisfaction of her appetites. Disillusions involved changes in character which, in another sense, were connected with her involution.

Woman Young, hernia L.V – S.I, herniatomy. Patient lived in a state of unconditional surrender to masturbation.

With this in mind, as well as for other reasons, a warning note was sounded at the Congress for Chiropractice (Freudenstadt 1956) against the chiropractical treatment of the manifest L.V – S.I syndrome and such treatment for hernia generally. When pregnancy is accompanied by a manifest L.V – S.I syndrome, we consider it necessary that the confinement should take place in a maternity ward under the supervision of an obstetrician. The patient's condition is precarious, in that she can scarcely be relieved of the cauda compression by an urgent operation, though, if the compression is prolonged, the disturbances in bladder function may either become irreversible or require a long time (six months to a year) to clear up.

"Caudal transverse lesions" at a high lumbar level (L.I – L.II and L.II – L.III) are also liable to bring on suddenly bladder disturbances which turn *out to be stubborn. Massive protrusion of a disc through a trauma or the* sudden herniation of a chronically diseased disc precipitated by an unfortunate movement produces disturbances in micturition – usually retention – which indicate urgent surgical intervention.

At the time of transdural herniatomy, chronic sphincter disturbances of bladder and rectum as also change and loss of sexual functions were among the not infrequent complications feared. One of the advantages of rhizotomy in the root sheath over the intradural method is that the likelihood of its involving any of these complications is remote. They no longer occur in their chronic form after extradural herniatomy. Nevertheless, 14% of the patients thus operated still show retentio (ad urinam), from which they are usually relieved within two days spontaneously or by means of appropriate medication (doryl carbaminoyl-choline chloride 12–25 mg). The vast majority of these patients suffer from L.V – S.I syndromes. During the operation it is possible *to tell whether a patient is going to be afflicted, transiently, with* bladder trouble (*e.g.*, after dissecting out S.1 and S.2 from adhesions, bud hernias under S.2, hernia from the corner between S.1 and S.2, etc)

We can chart the following possibilities according to our experience.

			Unilateral	*Bilateral*
Chronic lesion of total root	S.1		Bladder weakness?	Bladder weakness
Chronic lesion of total root	S.2		Incontinence	Incontinence
Irritation of root	S.1		Retention?	Retention
Irritation of root	S.2		Retention	Retention
Total rhizotomy	S.1	*in chronic phase*	No disturbances!	No disturbances!
Total rhizotomy	S.2	*in chronic phase*	Bladder weakness	Incontinence
Sensory rhizotomy	S.1	*in chronic phase*	No disturbances	No disturbances
Sensory rhizotomy	S.2	*in chronic phase*	Incontinence or bladder weakness	Incontinence

X-ray. Wide spinal canal, wide lumbar sac, spina bifida. Hernia nuclei pulposi. Herniatomy.

Trifling libido both pre- and post-operatively. In the first week of his marriage, patient had sexual intercourse once; later approximately once a month. Relations between husband and wife excellent.

Male (H) 28 years of age (po. 8458).

Somewhat debilitated, "sticky" character. Like 3 brothers and 1 sister, patient chronic sufferer from radicular pain and backache.

X-ray. Wide spinal canal, spina bifida L.V and S.I.

Nothing neurologically abnormal. Despite freedom from pain at intervals, increasing impotence and loss of libido since onset of symptoms. Coitus once in 3 months.

The following example will show how important it is to take the clinical record into account.

Male (L), age 24 (o. 14158).

Of athletic build. Normal psyche.

Five years previously: low back pain and radicular symptoms in the *left* leg; fruitless fusion operation. Since onset of symptoms, incontinentia ad urinam et faecum; subdued libido and potency, as transpired during his marriage.

Examination, neurological: L.5 syndrome on left.
myelographic: Recess L.IV – L.V.

Operation: Large hernia L IV – L.V on left.

In view of the clinical record (incontinence and sexual subnormality), disc L.V – S.I was also explored, right into the intervertebral canal for the S.2 root. On the *right* – hence contralaterally to the left-sided syndrome – this lay adhering to a paramedian bud hernia from the L.V – S.I disc.

Check-up (6 months): Patient free from pain, micturition and defaecation disorders cleared up, increased libido and potency; in patient's words: "normal again".

After having recorded the above cases, it may not be superfluous to emphasize the fact that we have received no complaints of obvious disturbances in sexuality after lumbo-sacral laminectomies performed on young people. Even the patient whose urine was retained for several days suffered no modification of his sexual powers at a later stage. Perhaps we might put it this way: The possibility cannot be ruled out that middle-aged male patients, whose potency was already waning or was still just normal, may experience a decided decline of sexual functions after manipulations to sacral roots.

Trophic disturbances

Incidence?

These should be understood as including the oedematous swellings on one side around the ankles and on the instep which may occur both pre-operatively through lesions and post-operatively after manipulations of roots. Their

Before the operation we knew nothing about the special conditions in the two latter cases. At check-up two months after the operation, both patients spontaneously asked about the possible reasons for the changes that had taken place in their sexual life. The previously existing inordinate drive towards sexual satisfaction had virtually disappeared.

Let us recall that lesions or irritation of higher sacral roots may induce incontinence or retention. Let us also remember that, as has often been noted, a certain structure of the lumbo-sacral spinal column (*e.g.*, spina bifida, with or without other degenerative features, such as a wide spinal canal, sacralisation and so on) is liable to be associated with incontinentia urinae. If an affection of certain caudal roots is able to inhibit the exercise of sexual functions – which has likewise been established beyond doubt –, [illegible]

How do the other psychic precipitating factors (disposition, education, conditions, moral standing) and the assumed somatic influence stand in relation to a hypersexuality that can legitimately be called pathological? To what extent are human sexual drives whipped up by a somatic condition to nymphomania, satyrism and other sexual aberrations?

The clinical history comes to mind of a previously healthy young girl, whose conduct had been irreproachable until she began to suffer from vaguely defined backache. As appeared later, to her own astonishment she began to feel in that same period a growing desire for sexual experience, simultaneously, her sexuality tended towards the homosexual, which alienated her from her home and involved her in social difficulties. The psychiatric diagnosis classed her as a psychopath. The circumstances mentioned came to light when she was put under observation for this backache, when it transpired radiologically that her spinal column exhibited those features which are regarded as signs of a degenerative back. The spina bifida occulta and the abnormally wide spinal canal were manifestations of that constitution which predisposes the subjects to incontinence, a disorder which, in some cases, yields reasonably well to surgical treatment.

It is legitimate to ask whether the same factors which brought on this young girl's backache also led to an irritable condition of the higher sacral roots. Their assumed sexual hyperexcitability could well have involved this psychopathic girl in the social difficulties described. The correlation between degenerative psyche, degenerative somatic manifestations and possibly sexual aberrations seems obvious.

We cannot yet venture to affirm that there is a causal relation between this ultimate and penultimate of the correlation, nor to tie up the evident therapeutic implications with it. If we could, then sacral laminectomy would be preferable to orchidectomy and women could therefore be relieved by the same therapeutic treatment as men.

We are aware of the combination of micturition disorders and declining sexual functions in sufferers from a degenerative back.

Male (H) 23 years of age (o 201151).

Character structure neurotic.

Before his marriage, patient seldom sought intercourse with girls, but was not impotent. Had had backache for years and radicular symptoms later.

once manifest eruption of psoriasis on the painful leg was cured in two months after extirpation of a hernia nuclei pulposi;

once cure of psoriasis on both legs within two months of injections into the hiatus sacralis (six times 20 cc. of 1% Procaine) for back complaints;

once sudden re-eruption of a cured psoriasis in the second week after the first hiatus sacralis injection for back complaints.

There is very probably a causal relationship here; it is incredible that these regressions and exacerbation should occur by chance in as chronic a malady as psoriasis.

Zukschwerdt (p. 171) also reports neurotrophic disturbances due to lesions of lumbar cauda roots (hyperkeratosis, clavi). On the analogy of periarthritis humero scaputilaris, a clinical picture is described – pain, limitation of function without demonstrable changes in the joints – under the name of "periarthritis coxae" given to it by Belart. Sympathetic, neurotrophic disturbances due to irritation of a lumbar root are held to be responsible for it.

Motor reinnervation after lesion of a motor root

This can be effected by

I. Recovery of the lost function.

II. Regeneration of injured nerve tissue, 1) through-growth;
2) sprouting of nerve rami.

The whole gamut of possiblities is known from practical experience. It will suffice, we think, to present them in the form of a chart. Each datum (on the left of the chart) may correspond to a recorded experience (middle column), while a probable explanation is suggested in the last column. It should be borne in mind, however, that the frequency of a combination is larger in proportion as data and recorded experience are closer together in the chart.

Anatomically verified	*Checked clinically*	*Explanation?*
No (visible) lesion.	Paresis clears up within 1 week.	Psychogenically restored function.
Partial lesion	Paresis clears up in 2–4 weeks	Restored function. Peripheral sprouting of intact tissue.
	Paresis clears up within 9 months.	Restored function. Peripheral sprouting. Fibre through-growth
Total lesion.	Paresis persists	Too much fibre injury

homolateral one-sidedness suggests a disturbance of trophic fibres in the injured root. They are by no means rarely noticed by the patient who has undergone a rhizotomy, both before and after mobilisation, and often disappear again two months later. They occur after the motor fibres have been injured; we have not noticed them after selective sensory rhizotomies. The swellings are occasionally accompanied by a "cold feeling", detectable both subjectively and objectively, but the clammy and cold feeling of one shank with pale or livid discolorations may also be experienced without swellings; sometimes, however, the patient is entirely unaware of this objectively ascertainable fact. Reischauer thinks that parasympathetic fibres in root L.5 may be affected; he considers that these signs reveal vasomotor disturbances pointing to a lesion of the L.5 root (paper read to the Congress of the Bavarian Society of Surgeons *in July 1956, p. 8). His postulate is that relaxation of the sympathetic antagonist* results in circulatory disturbances with dysbasia and intermittent claudication, with retention of the foot pulse, a usually normal arteriogram and a slightly or decidedly changed oscillogram, but always with a distincly lower temperature of the leg.

Reischauer (1949, p. 34) would seem to be contradicting himself to some extent when he says that no vegetative disturbances are caused by involvement of L.4, L.5 and S.1. We have seen trophic, vasomotor vegetative disturbances on one side in our patients after the lesion of one root, both of L.5 and of S.1. Seeing that the lowest sympathetic fibres pass *via* L 2 in their pre-gangliar course and the highest parasympathetic fibres (for prostate) *via* S.2 or, occasionally, *via* S.1, the anatomy of the autonomic system, in so far as it is known, does not offer a ready explanation of the above phenomena.

It is apt here to recall a *trophic disturbance* (that runs in families) *of the skin and the subcutaneous fatty tissue* around the ankles and instep The skin is cold to the touch in a band a hand's breadth wide around the ankles, though the remainder of the lower part of the leg and the feet are not cold The subcutaneous fat has increased, owing to which the cavities on either side of the Achilles tendon have filled up, persistent massage reduces the thicknesses temporarily. The accepted explanation is that fat accumulates due to a local subnormal temperature resulting from a minimum amount of trophic innervation. If the assumptions are correct, a local dystrophia of this kind would originate in an abnormal distribution of trophic fibres, hence in an endogenous variation of the cauda In the ultimate, therefore, the disorder could be said to be a manifestation of a degenerative back.

It is unlikely that derangement of trophic fibres caused by root lesions will lead to these permanent local dystrophias, for the spread of autonomic fibres is uncommonly wide, peripheral regeneration is rapid and permanent fibre casualties are soon compensated for by the coming into operation of the residual pathways.

As a matter of curiosity, we would record the following observations:

Psoriasis, diagnosed as such by the dermatologist, responded in three cases to interventions upon the cauda equina, *viz*,

It has been known for the past ten to fifteen years that innervation may take place, both in animals and human beings, in a third way (Edds, Wohlfart, see Woolf, Boone). As early as the second week after a partial lesion of the nerve, *fine collateral sprouts* are to be observed coming from intact motor nerve fibres. These sprouts are said to grow into the denervated muscle tissue and to make contact with the motor end-plates. As this growth begins in the second week, is massive (*i.e.*, according to circumstances, in many places simultaneously in the muscles) and the muscles are not yet atrophic (thanks to the function of residual nerve tissue and the brief duration of local denervation), this form of regeneration is said to result in comparatively rapid recovery of function, in several groups of muscles at the same time (2 to 4 weeks). This is *the period within which spontaneous recovery of a temporarily lost function is* improbable and regeneration due to through-growth over the continuity is still impossible anatomically.

Boone (p. 1061) considers fasciculations to be the result of hyperirritability due to reinnervation after preceding denervation. (See also Boone, Dissertation Utrecht, 1958.)

Empirically, fasciculations are encountered, if not exclusively, then at any rate predominantly in association with anterior horn lesions and, to less extent, with pathological changes in the anterior root. It would not conflict with this to associate fasciculations with peripheral nerve sprouting. It should be remembered that the healing of an anterior horn lesion is virtually ruled out; therefore, all reinnervation must derive from sprouting of motor nerve tissue that is still intact. Thus fasciculations in reinnervated areas after an anterior horn lesion must be due to sprouting. Reinnervation after a lesion of peripheral nerve tissue is effected by through-growth per continuitatem, or through sprouting. The more central the lesion is (anterior root), the greater is the likelihood that sprouting from neighbouring intact motor fibres has already taken place, before regeneration per continuitatem has been accomplished. The more plausible line of thought is, therefore, that sprouting is responsible for fasciculations when a peripheral motor lesion is far removed from the periphery (anterior root) and when the fasciculations occur long before regeneration per continuitatem could be effected.

We shall therefore leave undecided whether hyperirritability was meant of the peripheral sprouts, the newly reinnervated bundles of muscles or the anterior horn cells recently reunited with the periphery.

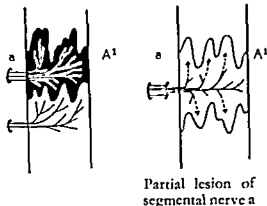
Electromyographically, fasciculations (bundles of spontaneous action potentials) are distinguishable from fibrillations (spontaneous single-action potentials characteristic of denervated muscle tissue). Boone observed fasciculations electromyographically in denervated muscle at periods when spontaneous recovery of function of anatomically uninjured nerve tissue was

As a matter of fact, we do know extreme cases of permanent paresis in association with roots showing no visible signs of injury and of complete recovery from a condition of weakness within one week of total rhizotomy.

The suggested explanations are founded on the results of experiments, muscle biopsy and electromyography, for which we refer to Woolf (Edds, Koelle and Wohlfart, see Woolf, p. 1506) and Boone (Edds, van Harreveld, etc., see Boone, p. 1061).

Muscle function depends on the complex of impulses (quantity and strength) reaching the muscle cell, an important point here being the Terminal Innervation Ratio, *i.e.*, the ratio of number of motor end-plates to number of innervating terminal nerve fibres. Assuming that nerve tissue regenerates, it will be evident that a muscle will not be provided with sufficient nerve tissue at one period but will at the next, so that the complex of impulses will be just enough at a given moment to effect a contraction. In this way seemingly miraculous cures can be brought about. Leaving the recovery from psychogenic paralyses out of account, it is equally clear why the *abatement of a functional disturbance* appears to take place from one day to the other. For, when the muscle again begins to function, there comes a moment when just enough impulses are again able to pass the interrupted nerve pathway (diminishing oedema? relieved compression?) to overstep the stimulus threshold in the muscle. Without some such mechanism, the sudden recovery of paretic muscles cannot be accounted for (such as sometimes after a trauma of a peripheral nerve, the dissecting out of a nervus ulnaris and the disappearance of a paresis after decompression of a root). A necessary condition is, however, that the peripheral muscle-nerve apparatus shall be intact, *i.e.*, that the terminal nerve rami and the motor end-plates are ready to function, waiting for enough impulses to get through to re-animate whole groups of muscles at the same time. Accordingly, we see rapid reinnervation taking place in entire groups of muscles simultaneously. As against this, there is the classical idea of reinnervation through regeneration, *viz.*, *the motor fibre* grows *via* a preformed pathway at the rate of about one mm a day, from the lesion right into the motor end-organ. This rate depends on the age of the patient and appears in practice also to be about right, or perhaps a little too low, for through-growth after a root lesion. Although the muscle is thus reached, in that phase contact has to be made with more or less remote end-plates, therefore, the recovery of function by reinnervation taking place in this way is gradual, bundle by bundle as it were. The recovery of function also depends on the condition of the muscle (atrophy), consequently, months may elapse between the moment when the fibres arrive and the group of muscles as a whole can contract powerfully.

Isomeric innervation of human, highly differentiated muscles, as Bolk sees it, can be represented diagrammatically. (Segmental nerve a; myotome A[1].)



Partial lesion of segmental nerve a

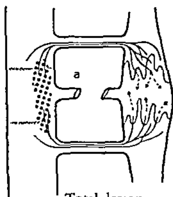
If a partial lesion has left only very few *a*-fibres, sprouting from these is likely to be slight.

How are we to understand the complete disappearance of a transient paresis behind a *totally* severed root in two to four weeks' time? (Restoration of physiological function is impossible and the interval for reinnervation per continuitatem too short.) The following are examples we know of personally:

Patient X (male), Nijmegen, L.5, peroneus musc.

Patient S (female), Wassenaar, S.1, peroneus musc.

The polymeric innervation of the muscles concerned was unable to prevent a *temporary* condition of weakness. Reinnervation must take place from the neighbouring segments. As far back as 1914, Quetsch (p. 123) presumed that regeneration of injured nerve fibres was effected in part by assumption of the function by fibres from other roots([1]). We have not come across this suggestion, or similar ones, in the literature since that date. We consider ingrowth of fibres alien to the segment in a denervated myotome to be improbable, as does Bolk. With Rabl's polymeric distribution of fibres in the motor root in mind, it seems to us that the most likely explanation for motor reinnervation under such circumstances is that it is effected by the entry into operation of a residual function (= activation of the motor transitional fibres already present), or by peripheral sprouting from motor transitional fibres.



Total lesion

History and Build-up of the "Transitional Fibres" Theory

The phenomenon of transitional fibres is closely bound up with the concept of segmentation and its morphological-functional consequences. The idea of segmentation has come to us through comparative anatomy and Bolk was among the first to construct charts of the segments of Vertebrata. The word segmentation in this context implies the building-up of vertebrate animals

improbable and regeneration per continuitatem not yet possible. He saw this in several cases of nuclear and peripheral partial lesions of nerve (including hernia nuclei pulposi) and attributed the phenomenon to reinnervation by sprouts from intact nerve fibres.

Hence we are again confronted with the concept of reinnervation by fibre through-growth (to be compared with sensory reinnervation, or overlap by through-growth according to Sherrington and Foerster). And it is incumbent upon us to ask ourselves again: Where do these sprouts come from?

Bolk was even more explicit about the motor innervation (from the same segment) of the muscles than concerning the sensory innervation of the skin, basing his pronouncements on his knowledge of anatomy and comparative anatomy. "During the first phases of development of the body, the boundaries between the myotomes are clearly visible, being separated by a thin layer of embryonic connective tissue" (p. 13). In the course of further development, this membrane continues to form the natural boundary of the myotomes (septum intersegmentale), or the original borders are entirely lost as the result of intensive differentiation. The septa intersegmentalia persist in fish and it has been established as a fact that the spinal nerve never extends its innervation area beyond a septum intersegmentale. "However intensively a myotome may differentiate itself (in man), the muscle material which has its origin in it is always connected with the isomeric (= the same segment) nerve" (p. 14). On page 5 Bolk writes: "Now, it is a peculiarity of the peripheral nervous system that it never plays an active part in the whole development of the vertebrate body; all through the course of development it remains passive, but also extremely conservative, in the sense that a segmental nerve does not exert a direct influence upon the differentiation of the area it innervates, but responds to the migration of any part it innervates simply by lengthening its fibres, or to extension and differentiation of its segmental area by further ramification. *Never, however, does it surrender any of its territory to a neighbouring segmental nerve or try to invade the latter's area*".

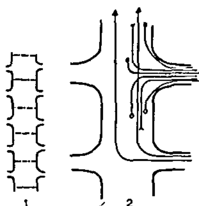
A more forthright statement on segmental-proprietary innervation of the myotome could barely have been made; it is a valuable pronouncement, resting as it does on massive anatomical knowledge.

The remark to the effect that the spinal nerve does not encroach beyond the clearly marked boundaries of the segment in fish evokes from us the comment that fish will in all probability have no transitional fibres in these clearly demarcated segments.

Bolk says the intercostal nerves in man are monomeric and the intercostal muscles monomerically innervated. Although we can adduce no proof, we suspect that the transitional fibres in the intercostal nerves of man will be few in number; the trifling overlap of the root areas would seem to point in that direction.

ruptedly into the spinal cord. This would mean that a lumbar segment of the spinal cord may extend to the cervical!

This conception is untenable in view of the high-grade differentiation of the central nervous system. If we set against this the artificial division found in textbooks and subjoined to "dermatome charts", (1) it will at once be apparent that such representations are mere working sketches which cannot bear any relation to reality.



In the light of these facts, it seems to us that the most acceptable *definition* is the following: Morphologically, the nearest approximation to a *segment of the spinal cord* is that part of the spinal cord situated opposite the root in which the majority of the incoming and outgoing fibres *via* this root terminate or issue; in a physiological sense a segment of the spinal cord is unlimited (2).

From this alone it follows that a root is multisegmental. We gained further insight into this by monoradicular mechanical irritation of the root in patients under a local anaesthetic. The connecting "isomeric" fibres (= of the same segment) between end-organ and spinal cord *via* roots of an "alien" segment we called *transitional fibres*, of which there are two categories, *viz.*,

1. Physiological transitional fibres, *i.e.*, a physiological distribution of fibres over the root, from which the root areas known to us empirically (root innervation patterns) result.
2. Anomalous transitional fibres, *i.e.* an otherwise than common distribution. The distinction is quantitative; morphologically, they bring about anomalies of the nervous system and, physiologically, variations in the functions of the roots, hence variations in their empirically established innervation patterns.

We find strong support for our view in the literature. Thus Bolk (1910, p. 28) established by dissection the connection by fibres of certain roots with uncommon innervation areas – uncommon, that is, by anatomical standards. (An example mentioned was a muscle belonging to root L.5 receiving its fibres *via* root L.4.) "An absolutely accurate segmental-anatomical localisation can never be made, as one never knows whether a patient's extremity is laid down, as one might say, higher or lower, or, to use a not very exact term favoured by English investigators, whether it is "prefixed" or "postfixed". A skin area which, with a high-fixed extremity, is innervated by the fifth segmental nerve, will, with a low-fixed extremity, receive its nerve fibres from

from common primary morphological units. In theory, each segment comprises a similar totality of organism and tissues supplied by the appurtenant part of the nervous system (isomeric innervation). Bolk defined this in 1910 (p. 3): Segmentation is a morphological subdivision, *i.e.*, the segments are the primary morphological components of the bodies of vertebrates. A somatic segment is a group of isomerically supplied tissues. By degrees, the sharply defined morphological boundaries between the segments have been more or less obscured by pronounced differentiation. An end-organ, such as the skin, should not be called "segmentally innervated", but an organ that is primarily built up in segments. "The principle of metamerism or segmentation is expressed independently of the nervous system ... The principle of segmentation is contained in the skin itself". The same vision we find in Starck, 1955, p. 334, quoting Detwiler, Hamburger and Weiss.

The phenomenon of transitional fibres is based on the isomeric innervation of a segment. And at this juncture we have to ask ourselves: What is a segment of the spinal cord? We have no answer to this question. There is no demonstrable morphological subdivision in man (in fish, segmental thicknesses of the spinal cord are still to be found). To refer to a grouping of motor anterior horn cells (Bolk, p. 10) in the nuclear columns of the muscle is merely to shift the problem to the question as to whether the spinal cord can be subdivided segmentally according to the accompanying roots. Cajal (and others) showed that afferent fibres descend and ascend in the spinal cord along several levels (*e.g.*, right into Goll and Burdach's nuclei). Lopez Cardoso (1937) and others describe the motor and sensory filamenta radicularia spreading extra- and intramedullarily across more than one level of the spinal cord in cats and dogs.

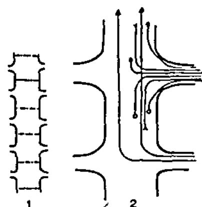
The more differentiated the organism becomes, the more do the divisions between the segments fade away. Partly for this reason, morphologico-functional intersegmental relations come to exist, which are manifested both intraspinally and extraspinally as follows:

An intensively developed internuncial system.
Long ascending and descending afferent systems.
Intersegmental outgoing and incoming filamenta radicularia (Lopez Cardoso)
Transitional fibres

The question therefore is whether one can legitimately say that the root determines the segment. For, if we adhere to the definition that the segment of a body is a group of isomerically supplied tissues, it follows that a root with the part of the spinal cord situated opposite to it belong to one and the same "segment" in so far as the afferent fibres, say, spread from this root uninter-

ruptedly into the spinal cord. This would mean that a lumbar segment of the spinal cord may extend to the cervical!

This conception is untenable in view of the high-grade differentiation of the central nervous system. If we set against this the artificial division found in textbooks and subjoined to "dermatome charts", (1) it will at once be apparent that such representations are mere working sketches which cannot bear any relation to reality.



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the sixth" (p. 29). In 1910, Rabl published his ideas of the polymeric composition of the motor root, in which views he was supported by Corning. Lopez Cardoso (1927) and others described the multisegmental composition of the filamenta radicularia. Brandt (1949, p. 496) states literally "that the posterior root is not segmented". Glees (p. 116) reviewed the opinions of Sherrington (1893—1898), Winkler (1902), Van Rijnberk and Ten Cate (1937) with reference to experiments on laboratory animals: the "dermatomes" behind a posterior root overlap and each section of a posterior root (filamentum radiculare) supplies the whole "dermatome". Foerster (1936) thought this could be observed in man. By measuring the evoked potential at the cerebral cortex, Kuhn (1953) established electrophysiologically in laboratory animals that every filament of a posterior root has a special function, either transmitting a given sensory modality more effectively, or supplying a certain part of a root area more intensively. On page 179 he writes: "Each dorsal root filament innervates but a limited portion of a dermatome. The cutaneous fields of the filaments are arranged within the dermatome in serial overlapping order". We have found this localising discrimination in man in the fibres of the posterior root right in front of the ganglion. Glees concludes his review with the remark that it is of practical use to be able, maybe, to explain certain dominating symptoms associated with disc herniation and root lesions. Well, there is abundant mention of these variations in the literature. (Davis, von Muralt and many others, see *loc. cit.* pages 285 and 380).

Thus the *multisegmental character* of the root - not only of the anomalous cauda equina, but also of the morphologically and physiologically apparently normal root system - became comprehensible, both morphologically and physiologically, on the evidence of the literature, with which our experience in practice fitted in (see p. 288).

The implications of this line of thought are various, *viz.*,

1. *In practice*

In 1910 Bolk predicted that "an absolutely accurate segmental-anatomical localisation can never be made" (p. 29).

2. *In the terminology*

One notes with astonishment that the classical investigators (Bolk, Foerster, Head, Sherrington, Ten Cate and Van Rijnberk) regarded the root as determining the segment and acted consistently on the definition that the root was monomeric; they therefore considered the end-organ innervated by it to be segmentally arranged and accordingly called it the dermatome, or myotome,

as the case might be. There can be no doubt but that these authors knew the fibre rising from a root into the spinal cord and descending, also the variations which were described in the Anglo-Saxon literature roundabout 1900 (Sherrington), as just so many end-organs "prefixed" or "postfixed" in relation to the nervous system (Eisler, 1892, see Norlén, p. 59). Bolk, who established the connection between root and end-organ by dissection – and used the root to determine the segment (p. 22) – was aware of this difficulty, as he speaks of "segment areas" (p. 8 for instance).

In modern clinical neurology, too, the root is used for determining the segment and reference is made to "dermatomes". From the point of view of medical practice, it is immaterial whether this term is exactly right or not, in so far as it is used to imply some connection between an innervation area found empirically and a possibly injured root. It is doubtful, however, whether the words "dermatome" and "myotome" are correct as applied to the innervation area of a root. The words "dermatome charts", etc. (*e.g.* Edinger, Keegan and others) do not cause confusion, because one knows what they mean, but actually they are incorrect. It would be better to speak of *root areas*.

3. *In principle*

The smaller anatomical "dermatomes" (according to Bolk) are not the same thing as the larger physiological "dermatomes" according to Foerster. This difference cannot be reconciled merely by referring to the different methods by which these "dermatomes" are determined. The difference is most clearly evident in the statements on the overlapping of two skin areas, or dermatomes, or root areas. When an end-organ, *e.g.*, a section of skin, appears to be supplied by two peripheral nerves, or two roots, or two "segmental" nerves, this is a physiological event established by exact methods which is called overlap. Bolk represents the opinion that a segmental nerve is confined to its own segment; it does not grow through other segments and does not admit any other segmental nerve to its own "domain" (p. 5). Bolk accounts for the "overlap" of dermatomes by intense differentiation of the segmentally laid-down skin: there are minute segmental skin areas side by side in the "overlap" areas, each supplied by a fibre of its own segment. "The dermatomes are partitioned by a zone which is innervated plurispinally". "Overlapping is not demonstrable anatomically; it is only the evidence of experimental investigation" (p. 115).





Foerster, Sherrington, etc. account for the excessive "overlap" of dermatomes by through-growth of fibres from one segment to another (*i.e.*, "alien" fibres). This seems inconsistent to us for two reasons, *viz.*,

a. It runs counter to the definition, *viz.*, that a segment of the body is a group of isomerically supplied tissues.

b. Through-growth of alien fibres also seems improbable, because it cannot under all circumstances account for certain generally known physiological phenomena, such as the reinnervation of sensory fibres. For instance, after the section of three posterior roots lying side by side, the outer two root areas are partially reinnervated. This reinnervation might be ascribed to through-growth of fibres from an alien segment of adjacent root areas. But why should this through-growth come to a standstill so that the central denervated root area is not reinnervated?

We know of an experiment (Nancy Miner, p 169), the results of which would seem to demolish the foregoing argument. "Unilateral excision of the three spinal ganglia of the hind-limb segments in frog tadpoles resulted in innervation of the affected limb by trunk fibers from nerves arising above and below the limb segments. Cutaneous localization in the affected limb was of normal character despite the atypical innervation"

We may add to this that a few months after the excision of a spinal ganglion (*e.g* for a perineural cyst) there were no demonstrable sensory disturbances.

Possibly this experiment might be explained by modification of the root area resulting from extirpation of the *ganglion*, meaning that this root area was "neutralised". The fibres in the adjacent root areas would then no longer be halted in their through-growth by defensive action on the part of the previously "alien" root area.

Detwiler's experiments afford some support for this suggestion. The addition of a somite to an ambystoma embryo generated an extraspinal ganglion plus nerve. The practical implications of these observations would be that extirpation of the sensory ganglion during sensory rhizotomy would foster reinnervation. We have, in fact, done this, but the evidence is not yet complete and cannot therefore usefully be recorded here.

If, however, we combine Bolk's view (innervations of a segment solely by material of one segment) with ours on the multisegmental composition of the root (the transitional fibres belonging to that segment in neighbouring roots), we do get a comprehensible explanation of numerous generally known physiological facts

In the foregoing we have tested this view in relation to several phenomena, in which the function which we ascribe to transitional fibres proved to be not unreasonable We recapitulate some of them:

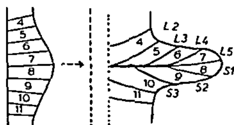
The division of the neural crests (*loc cit.* p 305).
The explanation of overlap (*loc cit* pp 314, 407)

Sensory reinnervation (*loc. cit.* p. 318).
Reflex changes and variations in the innervation of the muscles (*loc. cit.* p. 380).
Motor reinnervation (*loc. cit.* p. 399).
The residual function (deputising pathways in the central nervous system (*loc. cit.* pp. 322, 328)).
Antidromic conduction of impulses (*loc. cit.* p. 363).
Dissociated sensory disturbances though lesion of the posterior roots (*loc. cit.* p. 365).

Many other physiological facts which could be interpreted on the basis of the multisegmental nature of a root and of transitional fibres have not been discussed. For instance, why does an end-to-end sutured peripheral nerve not always grow out in the peripheral stump? Is this because the isomeric central and peripheral fibres are not placed one upon another? Again, why does a nerve transplantation from an alien segment fail, as, *e.g.*, the dissected out N.ulnaris on the peripheral stump of S.2?

We shall conclude this series with the addition of a striking example.

In embryological development, several parts of a segment appear to shift towards the terminals of the extremities (Bolk, p. 126). It was this fact that led to the well-known diagram in which segments 7 and 8 are expelled, as it were. The full line represents the partition between segments initially not contiguous, *viz.*, the differential line according to Bolk, or the longitudinal axis of the limb. It is likewise a fact that no overlapping takes place along – or rather over – this axial line (Biemond, p. 17, Foerster, p. 248, Kuhn, p. 180). (Droogleever Fortuyn holds a different opinion on this matter; p. 571) The same applies to the dorsal midline and, to less extent, to the ventral midline.



According to Sherrington and Foerster (and others), "overlapping" of the dermatomes is due to the spread of fibres into adjoining root areas. We quote Foerster again, p. 248. "Sherrington made it clear that the tactile dermatomes are more widely distributed than the pain dermatomes and that therefore a posterior root *penetrates with its pain fibres somewhat less deeply into the area of the neighbouring root* than with its tactile fibres".

Why is there no overlap across the axial line, say between segments 6 and 9, if overlap takes place by the invasion of an adjacent dermatome, *e.g.*, reciprocally between segments 6 and 9? The transitional fibre hypothesis would account for overlap between 6 and 7, 7 and 8, 8 and 9, etc. Regions 6 and 9 are

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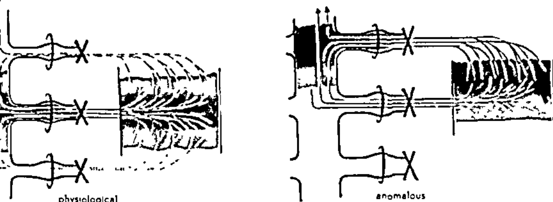
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The division of the neural crests (*loc cit* p 305).
The explanation of overlap (*loc cit* pp. 314, 407).



Transitional fibres.

sheath – or the intervertebral foramen – serves as a tube through which a large or small number of these fibres travels between spinal cord and end-organ.

12. On theoretical grounds, supported by embryological, anatomical and experimental facts recorded in the literature, a relationship may be assumed to exist between the distribution of the fibres in the roots, the division of the neural crest (in spinal ganglion) and the configuration of the vertebrae, especially the intervertebral foramina.

13. If the foregoing assumption is correct (12), it at the same time accounts for the frequent coincidence of an anomalous fibre distribution (anomalous transitional fibres, morphological anomalies, variations in function) with patent malformations of the spinal column, a concatenation which, in fact, is observed in practice.

14. Malformations of the spinal column are manifestations of what we call a degenerative back The coincidence noted in 13 emboldened us to consider the anomalous transitional fibres *themselves*, or their manifestation in morphologically ascertainable anomalies of the nervous system, or their manifestations in an anomalous or variable function of the nervous system, as evidence of a degenerative back.

Summary of Neurological Symptomatology

A summary of the neurological symptomatology of back complaints links the postulate formulated in point 14 with medical practice. We selected cases without morphological and functional changes and cases with obvious manifestations of the degenerative back. Which cases were and which were not to be regarded as such, was still a moot point. And this is where the comparatively doubtful value of the investigations comes in We shall revert to this presently.

neighbours by chance; segmentally, they are entirely unrelated, *i.e.*, transitional fibres have never existed between them; hence the absence of overlap. When segments 7 and 8 were expelled, the boundary areas and also the bordering nerve tissue, *i.e.*, the transitional fibres, likewise migrated to the periphery.

We have seen in this summary that transitional fibres manifested themselves in practice; that is to say, they became neurologically manifest. Sufficient evidence of their existence is to be adduced from anatomical, embryological and physiological facts recorded in the literature. Moreover, an interpretation of known physiological events on the basis of their implication proves to be not unreasonable. In view of what has been set forth in the preceding pages, we come to the following recapitulation:

1. The root is multisegmental.
2. Hence, the root does not define the segment.
3. What are called "dermatomes" in physiological parlance are, in fact, root areas.
4. The so-called "dermatome" determined peripherally to a root by dissection is a root area.
5. The so-called "dermatomes" defined empirically by neurologists are root areas.
6. The dermatome (or myotome, etc.) is isomeric by definition, *i.e.*, supplied by nerve tissue from *one* segment.
7. Root areas overlap because both motor and sensory roots are multisegmental.
8. Dermatomes (or myotomes) do not overlap, but, owing to vigorous differentiation, two end-organs, primarily laid down segmentally and permanently innervated isomerically, mingle intimately in the bordering regions.
9. Two dermatomes (or myotomes) determined by definition (or maybe theoretically) are incapable of overlapping. Under these circumstances, anatomical overlapping is a contradiction in terms and therefore does not make sense. It transpired that a boundary between two such dermatomes could not be demonstrated either anatomically or histologically. We decided that "overlap" does not exist anatomically and only appears to, physiologically.
10. Transitional fibres constitute the isomeric connection between end-organ and spinal cord, running *via* the (thus multisegmental) neighbouring roots. They are to be differentiated as physiological and anomalous transitional fibres, depending on the *quantity* of fibres which run in adjacent roots. Consequently, the segmental connection between end-organ and spinal cord can be represented by two diagrams.
11. The spinal roots are composed of motor and sensory fibres. The root

Love: impossible (*i.e.* 50%)
Young: impossible (*i.e.* 50%)

These are amazing figures, for, by simply "guessing" whether the level is L.V – S.I or L.IV – L.V, the chances of guessing correctly are fifty-fifty, or thereabouts.

We wonder whether the reason for the failure to obtain a correct level diagnosis in this group (about 35 %) taken from the world literature was that the patients were sufferers from an unrecognised so-called degenerative back. Let us deduct 10 from this percentage (for, after all, level diagnosis also proves to be fictitious in 10 % of cases with a "normal" back). In our experience, the remaining 25 % is quite an acceptable percentage for the group of persons with back complaints whose clinical neurological symptomatology, determined constitutionally, is out of the ordinary.

III. PSYCHO(PATHO) LOGICAL ASPECTS OF BACK DISORDERS

A.

In an appraisal of the interplay between the psyche and the sensation of pain, it may be useful to bear the following distinctions in mind:

a. Psychogenic pain. Apart from the question whether the intimated pain is really experienced by the patient, pain may be wholly or partly psychogenic in nature.

b. Interaction between psyche and pain. Emotion is known to be capable of worsening or subduing pain. Conversely, pain of evident organic origin affects the psyche of the sufferer. Pain can make a patient so irritable or overwrought that his behaviour suggests some degree of psychic disturbance. It may then be difficult to decide whether the pain was primary in relation to the psychic effect, or whether the psychological conditioning changed the reaction to pain.

c. Psychic predisposition to hypersensitivity to pain. A person's personality may be constitutionally so (degeneratively) constructed that a change in the perception of pain is adherent to the psychopathic or neurotic existence that he leads. Conversely, labile personalities of this kind are more likely to disintegrate under the influence of pain, even if this pain is of minor severity. Thus a vicious circle is created; the internal stresses set up make the condition of the sufferer appear to be insupportable, as the symptoms are unconsciously aggravated.

A tentative enquiry showed the proportions to be approximately 60 to 40 = 3 in 2.

150 "normal" backs: Level diagnosis on neurological signs	135 = 90%
100 obviously degenerative backs	60 = 60%
250 normal and degenerative backs	135 + 60 = 78%

The comparative value of these figures derives from, among other things, the approximately ascertained ratio of normal to degenerative backs, *viz.*, 60 to 40. This ratio would be quite different if *inconspicuous* manifestations of the degenerative back were included. We therefore took another group of 250 cases, this time unselected. This was the result:

250 unselected backs: Level diagnosis on neurological signs	70%
The ratio of normal to degenerative backs was then found to be 33:66	= 1 in 2.

Of little value – we repeat it – though these figures be, they would seem to suggest some provisional inferences, *viz.*,

1. As was to be expected, the number of inconspicuously degenerative backs is large.
2. Level diagnosis on neurological signs cannot be made with certainty per individual.
3. The chances of a correct level diagnosis, depending on neurological data, are as roughly 65% to 70% for an unselected case.
4. The chances of a correct level diagnosis on neurological signs rise in a selected case from about 60% to roughly 90%, in proportion as there are fewer signs of degeneration!

The uncertainty revealed by the above rough calculation of chances was likewise apparent in the reports published in the literature, to which reference was made on p. 284, *loc. cit.* and also in our own experience. Some degree of uncertainty in a correct level diagnosis has become increasingly apparent with the lengthening series of cases verified at operation.

In conclusion, we give the record abstracted from the literature by Ståhl (p. 142) with reference to the possibility of a correct neurological level diagnosis.

Correct level diagnosis of a radicular lesion, based on the clinical neurological picture (without radiograph or myelogram)

Ståhl	80%	
Notlén	70%	
Burns and Young	61%	Level diagnosis
Spurling and Grantham	50%	incorrect or impossible
Barr	50%	in about 35%

ical elements are not corrected, no amount of organic, mechanical or even surgical treatment will effect a cure". (Lewin, p. 641). "An analysis of data on surgically treated patients who reported a fair or poor result revealed that there was a massive functional component to the complaint" (Millikan, see Spiegel 1955, p. 345).

The association between the occurrence of psychic disturbances and back disorders is seldom related in the literature to the *total personality structure of the patient*, despite the fact that such a relationship might well be expected to exist, if only on the ground of familiar figures of speech in which the back appears as a part for the whole, such as "He has no backbone", or "It takes a strong back to bear such a burden", etc. Spurling (p. 93) emphasizes that "before operation is even suggested, there must be a careful evaluation of the patient as a whole with particular reference to his mental and emotional attitudes and his overall personality". The following quotation from Groen (1957, p. 51) – in which, as a matter of fact, the back is not mentioned specially – shows such a relation clearly: "Syndrome formation, suppression and shift, therefore, are phenomena of substitution, which develop under the influence of various factors of biological, somatic, psychological and social nature, which are all integrated in the *individual's total personality*". It is, moreover, more telling than the trite remark, made repeatedly, for instance, in "Psychosomatic Aspects of Surgery" by Cantor and Foxe, that the patient should be studied in his totality and not merely as the bearer of some or other local disorder.

Finally, we quote (in translation) some formulations by Hoelen (p. 191) which relate to this theme in its generality: "All organs may manifest a psychogenic functional disturbance". "A congenital peculiarity of the organs promotes the elective occurrence of psychogenic symptoms". ". . . .Owing to the dominating position of the nervous system, the neurologist is confronted with more psychogenic symptoms than the specialist in internal diseases . . .". "Only the neurologist who is trained in psychiatry can qualify as a reliable examiner". "The negative result of an examination by no means proves that a disorder is a psychogenic symptom. The probability that it is depends upon the finding of typical psychogenic somatic reactions during the examination, when the possibility should be borne in mind that this psychogenic reaction may derive from a latent organic disorder".

C. *The reflection of these views in some clinical data*

We collected 75 cases in which the patient retained symptoms post-operatively, ascertaining how many of these patients were held to be psychically disturbed.

A phenomenon known to psychosomatic medicine as *syndrome shift* occurs in association with psychogenically produced pain. Groen (1957, p. 33) writes: "Syndrome shift is the occurrence of a new symptom or syndrome consecutive to the disappearance of other manifestations of disease". Within the context of this phenomenon, organic syndromes, or psychogenic disturbances presenting as distinctly organic syndromes, are replaced by (*other*) psychogenic disturbances under the influence of intercurrent emotions. Quite conceivably, Leriche means the same thing when he says "une création continue de la douleur par la douleur", which means that if pain begins in one part, it will be felt elsewhere in a subsequent period – perhaps contralaterally.

In the matter of the reciprocity of the psyche and pain, it is interesting to note Rumke's views (Folia 1955, p. 277) on the transition between psychic health and psychic disease. He became convinced that there is no such thing as a smooth merging of health and illness. Health and disease have their own structure and their own "rules". He states further that, as against the higher and lower levels of integration in illness, it has to be assumed that there are steps in psychic health which are presumably subservient to phases of development. We consider that this view throws light on the atmosphere in which the dynamisms referred to under b. operate.

B.

Several references are made in the *literature* to the relationship between psychic symptoms and back disorders. Lewin (p. 622), for instance, devotes a chapter to the "Psychologic Aspects of Back Disorders" and Armstrong (p. IV) points out that, reluctant to part with former complaints, "patients . . . now gossip with some pride of their 'slipped disc'". Numerous authors emphasize the dependence of the operative results upon the psyche of the patient (Horvath, p. 46, Spurling, p. 93, Voris, p. 118, also see Spiegel 1955, p. 349). The latter writer insists that the operative prognosis of a compensation case is far worse and says in so many words that the prognosis of patients who have nothing whatever to do with insurance is considerably better.

We can easily elaborate this anthology. "What is the significance of the back in the body-plan of the patient? What is the patient's attitude to his back trouble? What does he really want when he visits his doctor? To be relieved of his disability or distress? Or does he want to hold on to it as an unpleasant but supportable means to a certain end? Does his back complaint serve as a mask for more or less unconscious imperfections?" (Veraguth, p. 214). "There is no section of the body to which psychosomatic impulses are directed more often and directly than the back This includes conscious and unconscious reactions". "The psychology of the person with a chronic back disorder must be understood in order to evaluate and correct his disorders". "If the psycholog-

Notwithstanding the lack of statistics, we propose to set forth the impressions we received in the clinic, our experiences and opinions relating to the occurrence of psychic disturbances in patients with low back pain (on the basis of degenerative back disorders), integrating these with the points enumerated in the preceding passages.

D.

When the diagnosis of hernia had become a commonplace in the clinic, it struck us from the outset that a large percentage of patients with low back pain as the result of a disc lesion-*cum*-herniation or other degenerative back disorders, proved to be psychically labile in varying degrees. Prick alluded to this fact as far back as 1943 at a meeting of neurosurgeons, on which occasion he expressed the opinion that the post-operative prognosis often depended on the patient's psychical condition before the operation. In the course of the next few years, this problem was explicitly and carefully investigated in every case of a disc lesion-*cum*-herniation and in all other cases of low back pain, both in the neurological and neurosurgical departments of the St. Canisius Hospital of Nijmegen (Director Prof. J. J. G. Prick), while in recent years this principle has been adopted in the St. Ursula Clinic of Wassenaar (Director Dr. Ed. Hoelen), where the matter has been studied.

For these investigations thorough, all-round medical examination was insisted on, with due regard for the heredity anamnesis, the developmental psychology and depth-psychology revealed in the clinical history; it included a multidimensional clinical psychiatric examination, in some cases supplemented by a testological examination. From all this, combined with an internal, neurological and neuro-radiological examination, it transpired that a significant correlation does exist between the neurovegetative symptoms (of the degenerative back disorders) and certain forms of the patient's psychic behaviour, deriving from labile integration.

We wish to make it clear at this point that this significant correlation was only plainly evident in the presence of symptoms, established either by anamnesis or clinical examination, which are commonly regarded as manifestations of a labile constitution.

Although these psychic changes in the various cases of the so-called degenerative back, far from being uniform, were, rather, considerably diversified, generally speaking the various psychopathological conditions had one point in common, *viz.*, the participation of an abnormal temperament in their production. To put this somewhat differently, we should say that if a large group of sufferers from degenerative back disorders are examined for their personality

"Recurrencies"	75	
Pre-operative diagnosis: Psychically disturbed	5	6.6%
Post-operative diagnosis: Psychically disturbed	*24*	*32 %*

These facts suggest the following:

1. Post-operatively, residual complaints were too readily entered as psychogenic.
2. Pre-operatively, patients' psyche was explored with insufficient attention.
3. In a number of cases, the retention of symptoms may well have changed the patient's psyche, so that a latent psychical disturbance became manifest as a reaction to pain and the disappointment experienced.

We went further and searched earlier clinical records to see how often "psychical disturbance" was diagnosed pre-operatively, *viz.*, in 300 cases, taken at random, of patients operated upon for hernia later, with varying results. This group, in which the examiner's attention was not specially drawn to the patients' psyche, was compared with 100 recent surgical cases in which the patients' psyche *was* studied with special interest before the operation.

300. Diagnosis "psychically disturbed", though the examination had not been specially directed to it	4%
100. Same diagnosis after examination specially directed	22%

The psychiatrically orientated examiner found that 22 of these persons had a character structure tending to neurosis or the psychopathic. When the facts had been collated, it was discovered that 18 of these 22 individuals presented obvious manifestations of a degenerative back (*viz.*, stigmata degenerationis, such as spina bifida occulta, transitional vertebra, wide spinal canal, etc.). Sixteen patients also exhibited vasolability, manifested by pre-shock without considerable loss of blood. Of the remaining 78 patients not having been diagnosed pre-operatively as being "psychically disturbed", only 19 (about 25 %) showed signs of degeneration in varying degrees; there was no appreciable vasolability during the operation in these cases.

Definite conclusions cannot be drawn from these meagre facts. To undertake the extensive psychiatric-psychological examination of a large number of patients and convert the evidence into statistics is a big task which it has not been possible so far to complete.

As a tentative test, five selected cases with obvious manifestations of a degenerative back were thoroughly explored psychiatrically All five were diagnosed as having clearly neurotic or psychopathic character structures and aggressive tendencies towards the outside world. Among five patients suffering from an uncomplicated hernia, without these manifestations of a degenerative back, there was only one case of a neurotic character structure with aggressive tendencies.

that of *neuropaths*. The latter are representatives of exudative diathesis; they are usually blond types with a transparent skin which, like the mucous membrane, is hypersensitive to infection and allergies. Moreover, the neuropathic condition as a psychic picture presents itself far sooner than constitutional nervousness; it can be diagnosed immediately after the birth of infants that are almost continually restive, have fits of screaming and do not fall asleep peacefully even after a satiating meal. A a later stage of development they often become stubborn thumb and finger suckers, then nail-biters, sleep-walkers and enuresis patients. In their kindergarten and elementary school years they seem to be bad eaters, eating their food slowly and reluctantly or not at all. Many display jactatio capitis as they fall asleep. In infancy they not infrequently have attacks of tetany or epilectic convulsions as equivalents of spasmophilia. Their passage through puberty and adolescence is usually difficult, with crises here and there. It is a remarkable fact, and a highly significant one within our context, that, at the time when these people begin to present symptoms of a degenerative back, they are no longer recognisable as neuropaths, but often appear to be psychopathic or neurotic and, upon careful examination, this condition proves to be the psychic manifestation of a psychosomatosis.

Therefore, if the somatic and psychic symptoms of a psychosomatosis are found in sufferers from degenerative back disorders, a searching enquiry should be made into records of neuropathic behaviour in early youth accompanied by signs of exudative diathesis, as also into the hereditary history.

However, the behaviour of the vast majority of psychically disturbed sufferers from degenerative back disorders is *neurotic* or *psychopathic*, or a little of both.

If one adheres to the view that both the abnormal psychic behaviour and the degenerative back with its consequent neurovegetative disorders are manifestations of a labile genotypical constitution, it follows that the conditions resembling neurosis cannot legitimately be termed pure neuroses. Further, since it is certain that the manifestations of a labile genotypical constitution are always the interference product of endogenous dispositions, on the one hand, and disturbing environmental influences impinging upon them, on the other, so that there can never be any question of an exclusive endogeny, it is evident that these cases are in no event pure psychopaths. Hence the *quasi* neurotic and *quasi* psychopathic conditions of sufferers from a constitutionally degenerative back are almost *invariably* due to unfavourable hereditary factors on the one side and appropriate disturbing environmental influences on the other.

We might add at this point that exactly the same can be said about these patients' physical complaints and symptoms which derive from their degenerative back. Here too there is the interplay between the adverse hereditary disposition and the adverse influences becoming operative at that particular

structure, this will be found to vary widely; but within this range of personalities one finds with striking regularity a pathological, distorted temperament which intensifies the sensitivity of all these different personalities to emotion-rousing conditions in their environment.

The evidence of the clinical material is that the forms of psychic disturbance in patients with low back pain due to a degenerative back range from constitutional nervousness, through forms of neuroses, psychopathic behaviour and psychosomatoses of various kinds, right down to epilepsy and mild psychotic conditions.

Before proceeding to comment in detail on these disorders, let us dwell for a moment upon their general symptoms.

One of the accompanying psychical syndromes found in sufferers from degenerative back disorders is, we said, *constitutional nervousness*. It is seldom difficult to recognise the picture of constitutional nervousness upon investigation into heredity, either in the ascendant or the descendant. A conspicuous fact emerging from this enquiry is that the other representatives of families with constitutional nervousness have also almost invariably been afflicted with back disorders of a degenerative nature.

Irritability is a typical feature of the psychic behaviour of these people. They are, moreover, emotionally unstable, "on edge", often uncontrollably impatient, especially at times when they are exposed to emotional stress, and are sometimes bad sleepers, in the sense of being unable to fall asleep at first, waking in the early hours and not sleeping peacefully until the late hours of the morning. As has been said, these people have an abnormal temperament, owing to which they are gloomy, either more or less persistently or episodically, and are prone, in a greater degree than their more balanced fellows, to suffer from the effects of all sorts of emotional situations. This last implies that to the outsider their psychically unstable behaviour often appears to be reactive.

At neurological examination, this category often shows signs of vegetative lability over and above the degenerative back symptoms. Such signs are dilated, strongly reacting pupils, symptoms of vasolability, a tendency to hyperhydrosis (arm-pits, palms of the hands, soles of the feet) and hyperreflexes without pathological reflex changes

Whereas the diagnoses "constitutional nervousness" and "neuropathic condition" are bandied about rather carelessly in the psychiatric literature, we have made a special point of enquiring into the possibility of distinguishing between these two at all. We say that they *are* distinguishable and would point out first of all that the type of constitution of sufferers from constitutional nervousness differs fundamentally from

this aggressiveness proves to be neurotically suppressed and distorted. The anamnesis of their daily lives shows patients in this category to be irritable, ill-adapted socially, troublesome, emotionally unstable; their neurotic restlessness and discontent, their inner tension and compulsions, oppress them; they complain frequently of headache, "a band across their head", gnawing pains in the head and, by no means least, of fatigue, lassitude and of feeling unequal to their social task. Many of them show signs of immature psychosexuality, some were driven to masturbation even in early youth, were harried by day-dreams of perverse sexual content, and sado-masochistic proclivities are also encountered in sufferers from degenerative back disorders. In a word, cases in this category present the symptoms of *physical* inferiority. It will be well in this connection to mention the various manifestations of the status dysrhaphicus, which will be described later.

Abnormalities of psychobiological behaviour correlate significantly with these physical imperfections. The various aspects of their *sensuous experience* have been unduly detached from the spirit, so that their perceptions and imagination are not tinged with the optimum human modulation. Their *instinctive behaviour* has remained too autonomous, it lacks an infusion of the spiritual and, as has been said, their sensual urges lack proper human control.

Besides this vegetative-physical imperfection, the miscarried development, the poor differentiation and shading of their animality, these people exhibit symptoms of arrested development in their *psycho-noetic* behaviour; they have failed to some extent to come to terms with themselves. That this faulty development entails failure in their *relationship with other human beings* will be plain and is a fact constantly confirmed in everyday experience.

Naturally, people who, through a labile constitution, have had to bear with an imperfect body from birth – one, moreover, which in a high degree disposes them to unstable psychic conduct and to fall short in social adjustment – are an easy prey to a sense of inferiority and to the *inferiority complexes* arising therefrom.

The clinical psychiatric examination within the framework of the testological [illegible] that the neurotic factors of a psychological and social nature are subdued in some sufferers from degenerative back disorders, while the inferiority complexes deriving from an organic physical inferiority predominate and are the foundation from which their neurosis develops.

Many back sufferers with pseudo-neuroses present symptoms of *hysterical conversion* at given moments in their lives. Psychically caused disorganisation of physical functions then occur, sometimes manifested in the motor innerva-

moment of life, through which the somatic aspect of the degenerative back becomes manifest.

Let us now first consider the *conditions simulating a neurosis* with which sufferers from degenerative back disorders are liable to be assailed.

Although we have been at pains to point out that these psychic disturbances are not neuroses in the true sense, *i.e.*, that they are exogenous yet partly conditioned endogenously, many of the features germane to the pure neuroses are found in these spurious ones. This is not surprising, since the endogenous disposition towards unstable behaviour realised through a pathologically developed temperament sensitizes the individual to environmental situations exciting emotion, and neuroticising factors in particular.

When sufferers from degenerative back disorders were examined by developmental and depth-psychological analysis, it was found that a diagnosis of neurosis seemed unavoidable, yet, on the basis of the degenerative endogenous factor, it had to be qualified as an impure neurosis.

Long before the symptoms of a degenerative back presented in these people, they showed signs of neurotic development. In their earliest youth they had been incapable of putting up any resistance to the effect of neuroticising influences in their environment, the disturbed temperament acting as a predisposing factor making them additionally vulnerable to the neurotic influences operative in youth and the con-

afflicted with an Oedipus complex in infancy, this serving as the foundation for their neurotic development. In this complex the repressing mechanism is revealed as the essential characteristic of the neurotic development.

When the "Ich" and "Überich" have been further realised in the later phase of development, many unconscious vital impulses (tendencies, propensities, drives, receptive needs) are rejected by the weak "Ich" and relegated to the subconscious Repressive affects operate in this repelling process of the "Ich", fear being the most common

From all this it will be clear why these sufferers from degenerative back disorders appear to be moody, not of a piece, insufficiently integrated in daily life and, socially, to be misfits, also why they are usually torn with anxiety, why they are perpetually taking a gloomy view of events and why their mood is predominantly pessimistic. Further, they are tortured by *conscious*, though unreasoning, fears, suffer from a vegetative reaction pattern of subconscious fears and their sleep is often disturbed by nightmarish anxiety dreams

The agressive behaviour of a large proportion of seemingly neurotic patients with low back pain is patently or disguisedly pathological. On closer analysis,

imitation of pathological bodily symptoms and the abnormal psychical behaviour that goes with them, is very rare indeed.

We now turn to the *pseudo-psychopathic* condition attacking sufferers from degenerative back disorders. We are aware that the neurotic or neurotic-like and psychopathic or psychopathic-like behaviours are by no means as clearly distinguishable as was originally thought. Combinations of the two groups of disturbances and intermediate forms of them are repeatedly being observed. Nevertheless, we think there is good reason to discuss separately the types of psychopathic behaviour displayed by sufferers from degenerative back disorders. It is, however, important to remember that neurotic dynamisms are almost invariably present in so-called psychopathic individuals and that those reacting neurotically repeatedly exhibit psychopathic idiosyncrasies.

The most striking feature of psychopathic behaviour is the lack of adjustment, especially social adjustment. This latter has in many cases a varying psychological foundation. The unadapted social behaviour of numerous back sufferers is conditioned primarily by a serious distortion of temperament.

It has been stated that we define temperament as the constitutionally determined kind of receptivity and reaction of the individual in conjunction with a specific vital ground-tone. The manner in which each individual lays himself open to the outside world and reacts to it is determined by his or her constitution. The individual's temperament determines his fundamental mood and also the permanent condition of tension preceding every real expression of life. Thus every individual has his or her own perception-tone, drive-tone and feeling-tone.

It is pertinent to the problem of the degenerative back that, from ancient times, a close correlation has been noted between a genotypically determined physical condition and the receptivity and reaction just referred to. This is evident from the classical names given to temperaments, such as sanguine, choleric, melancholic and phlegmatic.

Thus the *sanguine type* was said to be quickly responsive to stimuli, even mild ones, the whole of his psychic being responding, yet only briefly. His reactions are volatile, uneven and are externalised The stimuli take no lasting hold upon him.

The *choleric* responds quickly, the stimuli striking deep and engaging him for a long time. His reactions are fierce, comprehensive and explosive. High discharges of affect are constantly occurring. His ground-mood is aggressive and irate.

The *melancholy* type is slow to respond to stimuli, but, once the impressions are made, they strike deep and dwell with him. His reactions are tardy and very limited in extent His ground-mood tends to be gloomy. His power of retention is slow to discharge

tion areas affected by their degenerative back disorders. Such symptoms have something significant to tell us about the subconscious urge of the concrete individual. Sometimes they subserve the instinct of self-preservation, sometimes unsatisfied, passively receptive needs and they also on occasion serve as safety-valves.

There is a strong flavour of *narcissism* mixed up with the hystero-neurotic-like conditions just described. In such cases the patients are hypochondriacally preoccupied with their aches and pains. The affected parts of the body are an obsession of consciousness. Every extraneous emotional influence makes an imprint on the individual's attitude towards the imperfect part of his body. This naturally accounts for the fact that any intervention, especially a surgical one, sometimes produces effects contrary to those expected to result from the operation. It also explains why the operative prognosis of a certain category of patients with low back pain is *dubia ad infaustum*, to put it mildly. Indeed, if the psychic condition of the patient is not properly taken into account, so that surgery is resorted to following an incorrect indication, this may have adverse rather than favourable results.

Seen in this light, there is a sound explanation for a certain percentage of post-operative trouble. But it is very unfair to blame the labile psyche of the patient for *all* post-operative complaints and symptoms. Our investigation has made it clear that some of the post-operative complaints have an organic basis. It is even truer to say that there comes a moment in the lives of sufferers from degenerative back disorders with their associated psychic behaviour when objectively patent morbid physical symptoms are indissolubly bound up with abnormal psychic conduct.

It is a generally recognised fact, corroborated by research in the domains of psychosomatics and somatopsychology, that in every illness the somatic and psychical aspects are indissolubly intertwined. The more chronic cases of low back pain are no exception; the somatic abnormal element takes on the function of a pathobiological condition for an unadapted, unstable psychic form of behaviour. And the originally constitutional labile psyche, realised by the unstable temperament, manifests itself as a psychogenic superposition upon the bodily imperfection.

For this reason, no further explanation is needed for the fact that, through lack of inner poise, lack of homoiostasis, failure of integration and inner adjustment, people of labile personality are far more prone than the healthy to aggravate physical disorders and abnormal physical symptoms, and experience the associated psychical turmoil with pathological intensity.

In our opinion, *simulation*, *i.e.*, the conscious, rational, well-considered

siveness. This aggressiveness was discovered in a large percentage of patients, both at testological and clinical psychiatric examination.

We repeat that very many sufferers from a back disorder behave like vexed, emotionally unstable, easily irritated, uncontrollably aggressive persons who are incapable of adjusting themselves to the ever-changing circumstances of the outside world.

Although the intellectual stature of many sufferers from a degenerative back is anything but imposing, we cannot say that there were significantly more oligophrenetics among those we examined than in other comparable groups.

* * *

The endogenously (constitutionally) weak back need not in itself be an autonomous malady; it is, rather, one of the aspects of a more general disorder. By this latter we mean that a labile genotypical constitution which predisposes the subject to a spinal complaint and a radicular syndrome, at the same time holds the kernel from which a certain personality structure is liable to develop.

We have come to realise from our investigations and observations relating to endogenous back disorders with radicular symptoms in a large number of cases that the behaviour of such patients is psychically abnormal long before these spinal disorders become manifest. Different as the various psychic pictures might be, one characteristic was common to them all, namely a disturbed affectivity in the sense of the repression or tense control of feelings or passions.

We have now sought for some explanation that would account for the almost invariable coincidence of psychic maladjustment with spinal abnormalities. First of all we have to consider that, on the one hand a predisposition to an unstable personality structure, manifested by maladjusted behaviour, may possibly inhere in a certain degenerative genotypical constitution within the pattern of the chromosomes and, on the other hand, a proneness to develop a weak back. If so, this would be a question of linked heredity.

Another possibility is that the disposition to an existential design is anchored *in a labile genotypical constitution*, this design involving the actualisation of a disturbed affectivity, partly under the influence of unfavourable exogenous factors. In the sensitive-animal human being, this disturbed sensitivity may correlate with repressive dynamisms. Persistent repression of feeling and anxious control of affects put a person under inner emotional tension; that is to say, he or she is in a constant state of emotional congestion for lack of a normally human affective "exhaust". Internal emotional tension correlates

The *phlegmatic* type is poorly responsive to stimuli. The impressions he receives are faint. His reactions are slow and lethargic.

As has been said, the psychopathic behaviour of degenerative back sufferers is often to be understood as proceeding from a distortion of one of these types of temperament. We have seen many such sufferers whose mood was fundamentally depressive and whose affects were inadequately depressive, coming into play as reactions to certain situations of the environment fraught with emotion. In others, again, we found a pathological imperturbability or pronounced emotional instability in association with a more or less general asthenia. This asthenia, which we regarded as a defective ground-tone of the various expressions of life, manifested itself both somatically by an asthenic habit and by a fairly pronounced psychasthenic behaviour.

The disturbed temperament of people with a degenerative back was betrayed over and over again by a certain formula of emotional conflict, notably, an urgent need for loving care and attention, on the one hand, and a sense of emotional and social frustration, on the other. Quite often in this group of sufferers from a degenerative back the poor social adaptation was accompanied by adaptation diseases like gastric and duodenal ulcer, gastritis and other psychosomatic manifestations.

Besides exhibiting this psychopathic behaviour, which seemed to derive primarily from a distorted temperament, many of these sufferers conducted themselves more like character psychopaths. Their habitual patterns of action and reaction were maladjusted to the demands of society.

There were also psychopathic forms of drive behaviour in several cases of a degenerative back and there were likewise manifestations of endogenously enhanced sexual drives. In their youth, many of these patients had been addicted early to sexual perversions, such as masturbation and pseudo-masochistic behaviour. Later in life, matrimonial difficulties arose not infrequently, due to sexual incompatibilities. Such distorted sexual drives were very often combined with self-assertive and power drives.

It cannot but strike a qualified observer that many of these sufferers from back lesions have a feeling of inadequacy which, whether or not pathologically over-compensated for, results in conditions of social de-adaptation. One also comes across a pathologically heightened power-drive, which appears to transform the degenerative back patient into a despot within the family circle and in his social relations. The psychopathic behaviour which, therefore, is primarily manifested by symptoms of social de-adaptation or inadequate adaptation, is often determined largely by the results of uncontrolled aggres-

Let us suppose that 10 per cent. of a number of patients would derive benefit from a certain diagnostic intervention. The question then arises whether it is obligatory, or even permissible, to apply this treatment to the whole group of patients as a routine procedure. In other words, is it right, for the sake of introducing a certain intervention as a routine method, that 90% of these patients should undergo an unnecessary, possibly precarious treatment merely so that the 10% balance may have the benefit of it?

We think *not*. Here, as ever, the advantages have to be weighed against the drawbacks or distinct disadvantages possibly. Lipiodol myelography and thorotrast arteriography, more especially, would certainly have been condemned as inadmissible (routine) examinations if the dangers attaching to them had been known at the time.

In the following pages dealing with diagnostic intervention, therefore, particular attention will be paid to this relative value. It will again be seen how useful it is to have some knowledge of the patient's total personality when faced with this problem.

Lumbar Puncture and Lumbar Fluid Analysis

The composition of the fluid was known before the operation in 87% of our cases (see chart). Our records do not invariably show why this piece of information was lacking in the remaining 13%. In some cases myelography had been performed elsewhere without complete fluid analysis; in other cases, again, the lumbar fluid was mixed with blood and the data were therefore unreliable; if the patient was in great distress, the operation was not postponed for a repeat. If the composition of the fluid was not known after a lumbar puncture elsewhere (*e.g.*, for myelography), it was not considered opportune to waste time for the mere purpose of obtaining this information.

In one case only the *cells* had risen to 20:3 and on one other occasion to 8:3.

In 32.5% there were irregularities of the *curves* of the colloidal reactions in the cerebro-spinal fluid, which were either considered to be of no importance or were attributable to known causes, such as considerable increase in protein.

Of the fluids known, the *protein* content in 24% was higher than the normal 0.035‰, in 14% it was higher than 0.050‰. Seldom were contents higher than 1‰ found and the very high figures of 3 to 7‰ were among the rare exceptions. In such cases there was invariably total occlusion caused either by a tumour or by a hernia at a high lumbar level. The clinical history showed that the symptoms had usually existed for a considerable time.

No significant differences were found between the protein contents at

with physical tension in the sphere of the voluntary and involuntary musculature. The abnormal muscle tensions, which are the incarnation, as it were, of the repressed and rigidly controlled feelings and drives, bring about an abnormal posture and pathological motoricity. This pathological muscular contortion may eventually affect the supporting frame of the back so adversely as to produce morbid changes in the spine. And those organic changes may stand for the somatic substrate of spinal disorders.

Apart from this, it has to be borne in mind that the abnormal muscular tensions referred to, resulting in bodily steeling, abnormal posture and movement, may *per se* – i.e., without secondary organic changes in the supporting frame of the back – bring about spinal disorders and radicular pain.

The interrelation, if actualised, between a pathological mental state, as one of the manifestations of the labile personality, and its correlated muscular tensions, brings about a vicious circle implicitly involving exacerbation both of the spinal disorders and of the unhealthy emotional state.

Needless to say, similar dynamisms may occur in people who, subjected to exogenous neuroticising influences of a socio-affective order, pass through an upheaval which plays havoc with their development, and this is revealed by their neurotic and/or psychopathic behaviour. In such cases, too, there is an emotional tension finding expression in abnormal muscular tensions, resulting in spinal disorders with radicular pain, either deriving from an organic substrate or not.

Lastly, we must face the fact that if a genotypical constitution is responsible primarily for an endogenously weak back with its consequent signs and symptoms, a neurotic development may be superimposed upon it secondarily, causing inner emotional tension and its correlated muscular tensions.

IV. DIAGNOSTIC INTERVENTIONS

Some diagnostic interventions involve a certain amount of risk. It is right to consider whether it would be in the interests of all the patients to circumscribe a preliminary examination to the bare adequate minimum, without probing into the minutiae from which, as a whole, all the patients cannot possibly benefit. For instance, is a complete radiological examination, full spinal fluid analysis or myelography strictly necessary prior to an operation on the back? To decide one way or the other, therefore, it is well to compare the risks of diagnostic intervention, as far as the patient is concerned, with its value and the therapeutic consequences

of in advance. After all, it depends on the comprehensiveness of the anamnesis and thoroughness of the neurological examination whether indications be found pointing to the disorder recognised subsequently. We estimate that in about 1 % a lues cerebro spinalis, multiple sclerosis or some other disease was unexpectedly diagnosed. We did not once find a cauda tumour in our material without being forewarned by the composition of the fluid, which was by no means the case in departments where operations were performed on the back without a preliminary lumbar puncture. Further, very seldom did the detection of an exceptionally high protein content (such as 3 to 7‰) in our material prove to be a chance discovery, as, before the puncture, we had expected to find a considerable rise on the basis of the clinical data.

Whether a pre-operative lumbar puncture is necessary or desirable depends, to our mind, upon the thoroughness of the pre-operative clinical neurological examination; paradoxically enough, the more thorough this has been, the more pressingly does the examiner feel the need to check up his clinical findings against the composition of the lumbar fluid.

One of the *objections to lumbar puncture is undoubtedly the patient's aversion to it.* It involves psychological factors which have caused many a patient to declare, long after the event, that nothing they had been through, including the injection of the local anaesthetic and the operation under a local anaesthetic, had been as unpleasant as the pre-operative lumbar puncture. Much, of course, also depends on the skill of the person performing the puncture, but it causes more distress to some patients than others. About one in every twenty patients reacts to it more violently than is commonly the case and may suffer from the after-effects for as long as one to two weeks; the spontaneous sucking in of air and leakage of fluid through the perforation offer likely explanations of these after-effects.

Apart from these subjective objections, however, it must be admitted that lumbar puncture also entails some *risks*. The chance of *infection* subsequently is, we believe, negligible; we had no such complications in our material. There are several references in the literature to the danger of *disc puncturing*. Lewin (p. 81), Epstein (p. 363) and Spurling (p. 40) consider this danger to be anything but imaginary. The latter authors quote freely from the literature to show that quite a number of authors by no means minimise the danger of this puncture right into the disc and would even not rule out the possibility of a disc prolapse as the result. Lewin reports subsequent chronic backache in patients who had never complained about their backs before the puncture. Both he and Epstein knew a great many patients in whom the intervertebral disc was found to have narrowed at radiological check-up after a lumbar puncture.

lumbar puncture above and below a hernia nuclei pulposi at L.IV – L.V; but, in the case of a high lumbar hernia, the protein content of the fluid obtained from the lumbar region was found to be considerably above normal, in contradistinction to the normal composition of the fluid drawn sub-occipitally.

We do not know with certainty what causes the protein to increase in the fluid in association with hernia nuclei pulposi. It has been conjectured that protein is washed out in the fluid as the result, say, of local venous pressure. It would seem more likely that partial or total blockage of the fluid circulation at the site is responsible. But this suggestion is untenable in face of the fact that in 19% of the so-called "negative explorations (for hernia)" the protein content of the lumbar fluid was likewise found to have risen above 0.35‰.

The same datum was sought in cases of backs with undoubted signs of degeneration, enabling us to draw up the following chart:

	Protein content	
Higher than:	0.035‰	0.050‰
All back operations	24%	14%
Uncomplicated lumbar hernia	31%	
Uncomplicated high lumbar hernia		86%
Negative explorations	19%	
Degenerative back + hernia	28%	
Degenerative back + cauda anomaly	22%	
Merely degenerative back	20%	

The first fact which emerges from the above is that knowledge of the composition of the lumbar fluid provides no definite evidence of the existence of a hernia nuclei pulposi. Nevertheless, slightly increased protein does point to the probable existence of this lesion.

We have, moreover, definitely ascertained that in the 13% in which the composition of the lumbar fluid was not known, the lack of this knowledge did not affect the indication for the intervention, nor the way it proceeded, nor the post-operative course. Similarly, the result of the operation was unaffected by it.

It might rashly be concluded that lumbar puncture and complete analysis of the lumbar fluid are superfluous preliminaries to an operation on the back. But the pros and cons of this diagnostic procedure have to be considered. We therefore tried to ascertain in how many suspected cases of hernia nuclei pulposi lumbar puncture and fluid analysis led to the unexpected diagnosis of some other disorder. In this we failed, as it is impossible, after the event, to pin-point the cases in which the co-existence of another ailment was thought

had been cleared away. It might be compared with what is called a spontaneously covered perforation of the stomach. When we found such openings, we closed them with a tangential dura suture.

It has repeatedly been observed at explorations of the L.IV – L.V and L.V – S.I levels that, after some changes in the situation of the area of operation, clear fluid flowed from below the L.IV arch. In those cases the lumbar puncture had been made at the level of the L.III – L.IV disc. It is reasonable to suppose that a covered perforation re-opened after manipulation of the dural sac at the level of L.IV – L.V, causing leakage of fluid. We therefore think it is highly probable that various post-operative complications, taking the form of fluid cysts or temporarily aggravated serous secretion deep in the wound, are the result of leakages deriving from pre-operative lumbar puncture at a higher level. Consequently, we select the needle for intradural anaesthetization with great care, choosing one of the smallest possible calibre with a sharp point. As a precaution, moreover, we pierce the dura obliquely, so that the holes in the dural sac and arachnoid shall be as remote from each other as possible. In this way one membrane covers the perforation in the other.

To recapitulate what has gone before:

1. Analysis of the lumbar fluid does not provide positive data for the diagnosis of hernia nuclei pulposi.
2. In a small percentage of cases (approximately 1 %) the evidence of such an analysis points to the existence of disorders not previously suspected as the result of the neurological examination.
3. The disadvantages and possibly adverse consequences of lumbar puncture are trifling and, as they are usually due to faulty technique, can be avoided.

Some surgeons do not consider a pre-operative fluid analysis to be imperative as a routine procedure (see Spurling, p. 87). They argue, justly, that the neurological picture often points definitely to a lesion at L.IV – L.V or L.V – S.I to be treated by surgery, whatever the composition of the lumbar fluid might be. It certainly is true that the coincidence of some other disorder does not preclude the need for surgical treatment of an abnormality that has to be corrected in any event. We do, in fact, know luetic patients and sufferers from multiple sclerosis whose lumbar hernia served as the indication for surgical treatment. We also know one case of severe low backache and radicular pain in which the resumed examination after lumbar puncture led to the diagnosis of a silent high cauda tumour; after this had been extirpated, the patient retained his disabilities and had to undergo another operation later for a low lumbar hernia.

As against this, the composition of the fluid may continue to be upset for

One noteworthy fact is that the cases of most pronounced narrowing of a disc after lumbar puncture are invariably those of young people, mainly children. Does this suggest that the narrowing of that disc is due to the still fluid condition of the nucleus pulposus in youth, extruded through the perforation made by the needle? In a mechanical sense, it is barely conceivable that the tough tissue of an adult, healthy disc could protrude through as small a perforation as that caused by a lumbar needle. The key-words here, however, are "healthy disc", since a sufferer from backache is subjected to lumbar puncture precisely when he is suspected of having an abnormal disc. It seems to us that the prolapse of a disc through the perforation made by the needle is far more likely to occur if the disc is a degenerated one under some sort of tension. If taut ligaments over a prominence which is still just physiological are pricked by a needle, we do not deny that mucoid, degenerated nuclear tissue might issue through the hole. We have encountered something similar several times during an operation; with coagulating veins in the epidural space, it does unfortunately sometimes happen that the ligament on which these veins lie is also coagulated. Certainly, if such a ligament is under tension, it is liable to burst open and the interior of the intervertebral disc projects outwards to some extent. We have seen this so clearly, that we have accepted it as an indication for the extirpation of the damaged disc (to prevent further protrusion in the future), even if there had been no previous indication for clearing a no more than physiologically protruding disc.



Protrusion of a nerve root through a hole in the dura made by lumbar puncture.

Leakage after a lumbar puncture is a known complication for which the patient is managed by rest in bed, with the foot of the bed raised. After a lumbar puncture at the level of the area of operation – say at the L.IV – L.V disc – small holes have been noticed time and again during operations, with fluid issuing from them. These holes were seen to be covered with epidural tissue at first and the fluid did not begin to flow until this tissue

had been cleared away. It might be compared with what is called a spontaneously covered perforation of the stomach. When we found such openings, we closed them with a tangential dura suture.

It has repeatedly been observed at explorations of the L.IV – L.V and L.V – S.I levels that, after some changes in the situation of the area of operation, clear fluid flowed from below the L.IV arch. In those cases the lumbar puncture had been made at the level of the L.III – L.IV disc. It is reasonable to suppose that a covered perforation re-opened after manipulation of the dural sac at the level of L.IV – L.V, causing leakage of fluid. We therefore think it is highly probable that various post-operative complications, taking the form of fluid cysts or temporarily aggravated serous secretion deep in the wound, are the result of leakages deriving from pre-operative lumbar puncture at a higher level. Consequently, we select the needle for intradural anaesthetization with great care, choosing one of the smallest possible calibre with a sharp point. As a precaution, moreover, we pierce the dura obliquely, so that the holes in the dural sac and arachnoid shall be as remote from each other as possible. In this way one membrane covers the perforation in the other.

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1. Analysis of the lumbar fluid does not provide positive data for the diagnosis of hernia nuclei pulposi.
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As against this, the composition of the fluid may continue to be upset for

a long time after surgery upon the cauda equina and one can never be quite certain that a chronic change in this composition may not result from arachnoiditis, for example. In itself, this fact implies that the last examination before a laminectomy should be comprehensive for scientific reasons; hence the composition of the fluid must be analysed. Laminectomy is a major operation which should never be resorted to as a routine measure, as the result, especially of the first laminectomy, may be decisive for the patient's fitness and, therefore, for his happiness.

This is why we consider it undesirable – indeed, permissible only as an exception for sound reasons – to undertake an operation on the back without knowing the composition of the spinal fluid.

V. RADIOLOGICAL EXAMINATION OF THE LUMBAR SPINE *

This is not intended to be a chapter that could be fitted into a textbook on radiology. Our sole purpose is to enquire how radiology can help in the diagnosis of back disorders and in deciding on surgical intervention, or how it influences the surgical intervention itself.

Again we have to weigh the value of radiological examination against its disadvantages. Comparison would be most eloquently expressed by figures, but statistics are proverbially unreliable; and it is our impression that this is particularly so when the evidence of radiological examination is translated into statistical language. The enquiry is subject to the same objection that adheres to all statistical investigations, *viz.*, that the data collected in various departments under diverse conditions are not truly comparable. A second source of inaccuracy is the bias of the investigator, which, of course, applies just as much to matters outside radiology. However, the exact reproduction on the radiograph is a compelling monitor demanding a decision. In many cases the radiologist is required to pronounce upon it, but he can do no more than hint at "signs of". When such terms are resorted to, it means that the abnormality does in fact exist, whatever its clinical significance may be. The radiologist has to choose between digressing into descriptions of minutiae and overlooking inconspicuous changes, reporting the salient facts only. This is all very well in practice, especially if the radiologist should be constantly working with the same specialist, but it is a great impediment in the compilation of statistics. The subjective factor in the evaluation of "exact" data becomes

* Photographs from the radiological department, St. Ursula, Wassenaar, Head: Dr. E. Kruyff., formely Dr A van den Broek

plainly apparent when the same material is worked over at a later date. A passing symptom, *e.g.*, a positive reflex, is positive at that moment – and that is that. But if doubt is entertained as to whether there is a transitional vertebra or not, the matter can be discussed and, when it comes to arranging the material, the investigator can classify the case under whatever group his leanings prompt him to. In other words, the *danger of statistics in radiological investigation seems to reside precisely in its exactitude.*

Another source of inaccuracies in the statistical representation of radiological data is the *selection of the material.* Sometimes the *age* of the patient is significant; for instance, if the quest is for the relationship between congenital anomalies of the spine and, say, back disorders, it would be wrong to choose a group of young people as the field of enquiry. Thus the radiological examination of a group of young military recruits would provide no yardstick. If, by radiology, we want to compare the calcium content of a number of spinal columns statistically, we shall first have to place the patients into separate groups according to their ages. The decision as to whether a spinal column is rich or poor in calcium, as seen radiologically, depends on the subjectivity of the radiologist, the apparatus used, the hardness of the rays and the corpulence of the patient. The means of avoiding this subjectivity, *viz.*, the use of so-called calibrating plates, the constant permeability of which to X-rays can be compared with that of the spinal column, does not appear to be utilised in practice.

Selection may also intervene through categories of symptoms, from which the indication for radiological examination proceeded. We have seen statistics relating to the records of patients who were examined radiologically for an [illegible] spinal column also [illegible] were not made for the purpose of making the spine visible; someone suffering from an internal complaint may, of course, also have back trouble. Actually, however, individuals whose spine has been X-rayed by chance, are to be distinguished from those whose spine was deliberately X-rayed. In other words, the vast majority of these persons suffering from an internal disease had no back complaints. Consequently, these records do not provide the proper material for establishing the incidence of complaints and symptoms in an entire group of the population.

Lastly, the *specialist* referring the patient for radiological examination brings a strong flavour of selection into the matter. This is obvious where patients with backache are sent by the specialist for internal diseases, the gynaecologist or the orthopaedist. In any comparison between these different groups it would be necessary to allow for the fact that the specialist in internal diseases, for example, will generally have far fewer patients X-rayed for back disorders than

the orthopaedic surgeon and the statistics would have to be modified accordingly. The statistics of an orthopaedic department showing the incidence of abnormalities on radiographs will be different in their arrangement from those compiled by a neurological department. Similarly, the radiological statistics relating to patients sent primarily to the neurologist will contrast noticeably with those pertaining to patients referred in the first place to the neurosurgeon. Both on the part of the referring physician and of the patient there is a certain amount of selection between these two specialisms. Generally speaking, there will be a tendency to refer dubious syndromes for diagnosis and treatment to the neurologist, who, as a rule, tends towards the conservative; by contrast, the majority of patients sent to the neurosurgeon have already been treated conservatively elsewhere, or their disorders were so manifest that direct reference to the surgeon was preferred. On account of this selection, a far larger number of obvious cases will appear in the neurosurgeon's material.

The statistics which follow are submitted with all the reservations implied by the above considerations. Our only reason for submitting them at all is to be able to compare them with statistics given in the literature. In spite of their ineluctable imperfections, we shall try to establish:

The incidence of radiological indications of a hernia;

The incidence of radiologically visible signs of degeneration;

The frequency of a causal relation between radiological changes and back disorders.

These figures relate to a mixed batch of neurological and neurosurgical patients; all the patients, with but few exceptions, were X-rayed on account of back disorders or radicular pain. The statistics, given as a percentage, were drawn up with reference to examination in groups of hundreds of persons and for this purpose we had the records of upwards of 10,000 individuals.

Radiographs without Contrast Medium

Plane radiographs in antero-posterior and lateral projection are to be taken as complementary components of a routine examination. The following were visible on these radiographs:

Congenital disorders	Spina bifida occulta	Manifest	9%
		Indicated	8%
		Not revealed radiographically, but observed at operation	2%
	Pseudo-spina bif. occ.		5%
		ditto	4%

	Transitional vertebra	Manifest	*ca.* 7%
		Indicated	*ca.* 35%
	Spondylolysis		(unassessable)
	Spondylolisthesis		4%
	Wide lumbosacral spinal canal	Manifest	7%
	(difficult to judge)	Indicated	25%
Residual conditions from traumata:			< 1%
Other acquired morbid changes:			
	Spondylarthrosis generally	Age > 50	60%
		> 40	50%
		> 30	20–30%
	local		17%
	local together with hernia		20%
	Posterior lipping	Manifest	9%
		Indicated	9%
	Indented end-plates		see p. 446
	Disc narrowing without hernia		see p. 460
	Disc narrowing with hernia		see p. 461
	Pseudo-spondylolisthesis		6%
	Functional listhesis		7%
Semi-lateral projections:	Spondylolysis		
	(in about 25% on semilateral projections)		*ca.* 4%
	Spondylolisthesis (hence also 25%)		4%
	Detached arch (rare)		
	[illegible]		7–25%
Function examination:	[illegible] + 6 + 7) =		17%
Special projection	Small angle sacrum – spinal column		
	(tilting of L.V vertebral body)		
	(opening between S.I and S.II arches)		
	Sacral foramina		
	Thoracic and cervical exposures for counting transitional vertebrae		
	Lumbo-sacral details		
	Radiographs of pelvis (hip)		

Hence an X-ray examination before an operation for back disorders, intended to provide complete orientation, involves approximately nine exposures, to which are to be added post-operatively two exposures for level control (clip, silver threads along roots), exclusion of vertebral slipping through discotomy, etc.

Most of the details listed in this chart are self-explanatory and several abnormalities were discussed in the relevant literature survey. Some parts call for further discussion.

Pseudo-spina bifida occulta is the name for an opening between two sacral



Pseudo-spina bifida occulta S I – S II

arches, usually S.I and S.II. This is visible at operation in 4 % more cases than on radiographs (5 %), which is due to projection of the exposures. Recognition of the defect is useful in determining the L.V – S.I level during laminectomy, during which the tendency is to go by the lowest palpable opening between two arches (*i.e.*, L.V – S.I). Without utilising radiological indications, there is a 9 % chance of determining a wrong level; with their use, there is still a 4 % chance, which can only be avoided by manually seeking the level of the pelvic crests and comparing this with the level of the intervertebral L.IV – L.V disc on the radiographs. As a rule, the L.IV – L.V disc is situated 1 cm below the connecting line of the pelvic crests.

We regard the opening between S.I and S.II as the manifestation of a *subclinical tendency towards lumbarisation*. It may be the only manifestation, but we have repeatedly seen a coincident transitional form of the bony spinal canal at the L.V – S.I level; the pedicles of the L.V and S.I arches fan out and thus cover the roots in the lateral recess, just in front of the foramen; the spinal canal is a flattened oval, like the foramina; the lamina between the intervertebral canal for the S.1 and S.2 roots reaches further in a cranial direction. In our opinion the recognition of this condition is an indication for facectomy and canalotomy (see p. 606).

Vertebral slipping * was observed in 17 %. This figure differs from the statistical records given in the literature (manifest spondylolisthesis in the total population roughly 2 %, in orthopaedic and neurological-neurosurgical practice approximately 6 %). Our 17 % includes those cases in which the radiologist reports "signs of some degree of slipping". These cases also call for attention, as the patient's distress, or lack of it, does not always keep step with the degree of listhesis; indeed, it is our impression that the dislocation associated with incipient listhesis may cause more distress than a severe established one.

True *spondylolisthesis* is a vertebral displacement due to either a traumatic or congenital lysis in the arch (4 %). When we tried to find a lysis in association with a displacement, by having semi-lateral films made, we found it in about 25 %.

* Records in the literature were checked in collaboration with Dr. H. B. Goettsch with reference to radiological material from the St. Joseph Hospital at Tilburg, Director Dr J. B Stolte



In the literature (Junghanns), *pseudo-spondylolisthesis* is the term given to displacement resulting from the narrowing of a disc with the small vertebral joints directed semi-obliquely (not vertically). Through this narrowing, the vertebral bodies approach each other but, owing to the slant of the joints, the upper vertebra slips, usually backwards in relation to the lower one (6%). As the arch remains intact, the anomaly is referred to as a pseudo-spondylolisthesis.

Besides these two forms, we came across displacements with the arch intact and the disc not narrowed. This might be called a "pseudo-pseudo-spondylolisthesis". In one quarter of the cases these displacements were to be seen at more than one level. We calculated that this kind of displacement occurs in 7% of neurological-neurosurgical patients and consider that it accounts for the frequency with which displacement in varying degrees is mentioned in radiographic descriptions.

Closer analysis of these displacements strongly suggests that they are due to mechanisms which, in our view, account for many vaguely defined back disorders.

The total mobility of the spinal column is subserved by the co-ordination of all mobile parts. Junghanns (see Schmorl, p. 23) speaks in this sense of "Bewegungssegmente"; he states with emphasis that "the joint of the head and the way the pelvis and lower extremities are able to carry the body are not without their influence upon the function of the spinal column". A congenital or acquired defect of one "mobile segment" acts upon another or several others

We have noted that in cases of displacement without lysis in the arch or narrowing of the disc involved, abnormalities almost invariably existed at levels other than that of the detected displacement. Mobility is checked by the existing abnormality at this one particular level; for the total mobility of the spinal column to be maintained, the movements at other lumbar levels have to be performed with greater amplitude. Possibly this compensation itself leads to local strain, local symptoms, degeneration and narrowing of the disc, with pseudo-listhesis as the result if the position of the vertebral joints favours it. If, however, the other lumbar levels are unable to compensate adequately by increasing the amplitude of their normal movements, either the total mobility of the spinal column will diminish (locking, neutralised lumbar lordosis), or it will be forced in its motile parts. This locking is effected by muscle spasm, depending on the condition of the ligaments, and such muscular



Pseudo-spina bifida occulta S.I – S II

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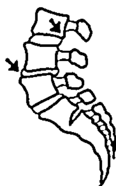
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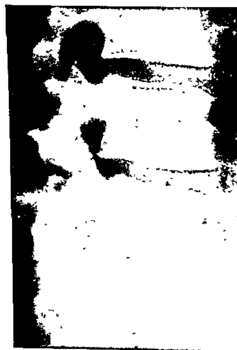
spine then, it seems to us, goes to show that the functional listhesis must have been primary.

As was to be expected, functional listhesis (7%) proved to be more general and have a more subclinical aspect than pseudo-listhesis (6%). With a simultaneous hernia, we more often found this in the pseudo-form at the level of the displacement, whereas, in association with functional listhesis, it was usually at a higher level. Sometimes we found the two forms combined; hence, mutatis mutandis, the whole mechanism described above came into play.



Combination of pseudo-spondylolisthesis with a functional listhesis (rudimentary S.I – S.II disc).

There is extensive literature on true and apparent retroposition (or, as the case may be, anteposition) of vertebrae. Some ascribe the apparent form to considerable disparity in antero-posterior dimensions of the vertebrae (Garland, p. 290, and Hagelstam, p. 93). Other causes mentioned are the concavity of the posterior aspect of the sacrum and other anomalies (Melamed and his colleagues, pp 318 and 327). (c.f. Guntz, Mulder, Weber).



Congenital anomaly.
Pseudo-retroposition
Small antero-posterior dimension of L V



Functional listhesis? Slightly degenerated disc. Osteoporotic spine (Woman, aged 42)

tension causes fatigue and, in the long run, pain. Any abnormality which locally checks the mobility of the lumbar spine, or causes pathological motor action which has to be compensated for by muscle spasm, may be enough to produce backache, the more so if the muscles of the back are not in good condition (asthenia, dystrophia musculorum progressiva, after an extensive laminectomy). If efforts to keep the back straight are abandoned, the result is *hyperlordosis. If, through a local lesion, the spine is unable to realise the* curvature of this hyperlordosis, the lumbar vertebral column is forced elsewhere. The disc and joints are jerked, twisted and dislocated until, to acquire the necessary curvature (whether hyperlordotic or not), the vertebrae begin to slip relatively to each other. A displacement of this kind is the result of adjustment to the function of the spine and is therefore a *functional listhesis*, a



Functional listhesis with relieved lordosis, following trauma of the upper lumbar region



Functional listhesis
Long sacrum



Functional listhesis
Hyperlordotic spine

better name for it, we think, than torso-listhesis, luxation-listhesis or rotation-listhesis. Let it be stressed again that the arch remains intact and the disc in between does not narrow, at least, not to begin with. If the tissue of this disc degenerates, a disc lesion may ensue secondarily, with narrowing and, possibly, herniation The functional listhesis may then become the pseudo-listhesis described by Junghanns, depending on the direction in which the vertebral joints point; once this pseudo-listhesis has become established, it is no longer possible to discover which was primary, the displacement or the narrowing of the disc. The existence of movement-impeding factors elsewhere in the lumbar

We have tried to track down the factors inducing *functional* listhesis and these are what we have come to:

Trauma at the thoraco-lumbar level.
Hernia at a higher level (*e.g.*, L.II – L.III).
Synostosis (congenital)
(acquired: fusion!!)
(acquired: *e.g.*, after discectomy!)
(acquired: through infection).
A long sacrum (*e.g.*, *transitional vertebrae*).
Neutralised lordosis (= functional "synostosis").
Hyperlordosis associated with:
pregnancy
asthenia
dystrophia musculorum progressiva.



Functional listhesis
Sacral cyst
Transitional spine
(long sacrum)



Trauma L.II – L.III
Functional listhesis L.V – S I
(long sacrum)
(transitional spine)

Hagelstam (pp. 110–115) shows, advancing arguments well supported by statistics, that "there is no bony structure barring a vertebra from gliding backwards.. " "The appearance of retroposition is not dependent on the position of the planes of the intervertebral joints only" (p. 116).

Knutsson stresses the instability of the spinal column and therefore considers retroposition to be "a temporary position, a phase in the movement". Hagelstam (pp. 16, 98, 116, 118, etc.) refers to many authors who consider instability and retroposition as being caused solely by degeneration of the disc *at the level* of the retroposition. On page 98 he says: "Simons states that discovery of a retroposition permits of the conclusion that the disc is degenerated even though the height of the disc is not reduced". Hagelstam (p. 107) gives examples of retroposition around a disc of normal height (backward-*tilting* L V over L IV after cured spondylitis tuberculosa L IV), presuming that this disc had nevertheless degenerated as the result of the tuberculous process. Looked at in this way, all these forms of retroposition *around* a degenerated disc are nothing but special aspects of Junghanns' pseudospondylolisthesis. What we are particularly concerned with, however, is retroposition *without* known degeneration of the disc, especially that resulting from faulty movements owing to morbid changes at other levels of the lumbar spine.



Multiple functional listhesis
Fracture due to compression
L II – L III
Osteoporotic spine
(Man, aged 12)



Functional listhesis,
asthenia

Tenaciously neutralised lumbar lordosis is, as it were, a functionally caused "synostosis". If one part of the mobile spine is locked, some other part has to compensate for this local rigidity during movements. In this way a local rigidity, manifested as a neutralised lordosis, may be the cause of a functional listhesis; from which it follows that every disc lesion may be the cause of functional listhesis.



Multiple functional listhesis. Hyperlordotic spine

Multiple functional listhesis (rudimentary disc S I–S.II in long sacrum)

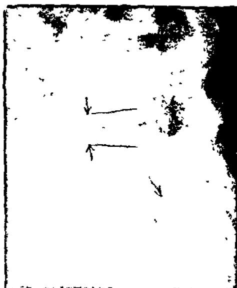
At the same time the danger can be glimpsed of a pathological motoricity resulting from, let us say, an untreated disc lesion, or one wrongly treated by conservative methods (such as rest). It transpired that laminectomy (with or without spondylodesis) was most frequently the cause of the occurrence of a functional listhesis. If, with this in mind, one makes appropriate enquiries, one finds that, if low back pain has only temporarily ceased after a laminectomy, it usually returns two or three months later. In our opinion this is due to insufficient tonicity of the back muscles during adjustment in the post-operative period; on the other hand, this backache is due to failure of the spine to "take



Hernia L II – L III
Pseudo-spondylolisthesis L.II – L.III
Functional listhesis L.III – L.IV



Hernia L.III – L.IV
Functional listhesis L.V – S I



Hernia L IV – L V
Functional listhesis L V – S I



Hernia L V – S I
Pseudo-spondylolisthesis L V – S I
Functional listhesis L.IV – L.V

will bring about real compression of the root, with radicular symptoms as the result. As we know, there is a high incidence of false positive and false negative myelograms in a flat wide vertebral canal and also of false positives in retroposition of vertebrae. Possibly a few of these cases (3 %?) account for some of the so-called "negative explorations" for hernia.

Local spondylarthrosis around a disc (17 %) is a sure sign of a chronic process in that disc. Small hooks on the anterior aspect of the vertebral body are seen in the lateral lumbar plane radiographs. If further examination does not provide convincing localising clues, one can confidently attack the level corresponding to this local spondylosis at exploration. In this we may be guided by the assumption that the pathological motoricity around a local process produced this local formation of hooks. The percentage reported agrees fairly well with the incidence (15 %–20 %) Krayenbühl found for hernia accompanied by local spondylarthrosis.



Local spondylarthrosis



Posterior lipping

Posterior lipping was demonstrable definitely in 9 % and probably in 9 %. Reischauer (p. 37) pointed out that posterior lipping does not appear on radiographs as frequently as it actually occurs. (Hagelstam, p. 82, 2 %; Solomon, 3 %; Briggs and Krause, 1945.) The lower rim of the vertebral body above a

the bend" round the artificially made synostosis without forcing other mobile segments. In patients with an adequate muscular system this leads to temporary or permanent post-operative rigidity (with fatigue and low back pain); sometimes a special kind of motoricity is developed, when, after stooping, the patient "works" the spine into the upright position, as with true spondylolisthesis. In sporadic cases, a patient with such tendencies will convert his residual symptoms (which are real enough anyway) into an hysterical exaggeration of this special motoricity, so that it becomes convincingly psychopathological. We then call it an hysterical corkscrew phenomenon.

The following considerations regarding functional listhesis do, we feel, also come within our context. Every displacement causes a relative limitation of space within the vertebral canal, without necessarily causing distress or symptoms. This depends on the degree of displacement, certainly, but also upon the available space in the canal, therefore upon the *constitution* or condition. Many such conditions are known (arthritis, osteochondritis, posterior lipping, and so forth). One such type of constitution would be Verbiest's stenotic canal, but, as far as we are aware, this has not yet been encountered combined with a listhesis. Any constitutional relative stenosis would qualify, particularly the flat sacral shape of the lumbar vertebral canal associated with transitional vertebrae, but also the flat oval vertebral canal in general. Even if there are no other visible signs of transition, the wide but flat oval canal is to be regarded as the manifestation of a sacralisation tendency. A flat, wide lumbo-sacral canal of this kind is not the same as the canals, also dorso-ventrally wide, which were described by Elsberg and Dyke in the thoracic regions and by Jefferson Jr. in a high lumbar region. Maybe the combination with spina bifida described by Walker and others should be included, though they did not mention a predisposition to stenosis in their descriptions. It is our view that the flat, wide lumbo-sacral vertebral canal is the manifestation of a type of constitution which predisposes to relative stenosis.

The incidence of the flat, wide canal in neurological/neurosurgical patient material is between 7% and 25%, for the obvious and subclinical forms respectively. Functional listhesis occurs in roughly 7%. The combination of functional listhesis with a flat, wide lumbo-sacral canal might, therefore, be expected to occur in 7 . 4 = 1¾% Their coincidence, however, turns out to be something above 3%, which is twice as much almost. Are we to infer from this that, in itself, the spine with a flat, wide canal predisposes to functional listhesis, possibly owing to instability?

When this type of constitution is combined with functional listhesis, the chances are that the two factors predisposing to stenosis, acting in conjunction,

under 50, one may gather that the tissue in the disc at the site is degenerating; the same is true in a patient past 50, but the symptom loses its value as an indication of pathological change in the disc.

With due allowance for the subjectivity of our assessment, we come to the following round figures:

Patients (under 50)	*Indented end-plates*
Without back disorders	approx. 5%
With back disorders	approx. 17%
With narrowing of the disc	approx. 33%
With verified hernia	approx. 50%
With posterior lipping	approx. 75%

Reduced or neutralised lumbar lordosis is not infrequently taken to indicate the organic genesis of back disorders. It is sometimes found by chance during examination of the gall-bladder and stomach or for indeterminate abdominal complaints. We have to consider whether perhaps the symptoms for which the radiological examination was undertaken were the result of general asthenia which at the same time brought about weakness of the muscles of the back and the change in the lumbar lordosis.

We would recall that, as stated elsewhere (p. 56), reduced lumbar lordosis is to be regarded as a defensive mechanism against painful anomalies of posture and movement. This mechanism can be maintained for as long as the muscles involved can hold out; clearly, even if the muscles get tired, the patient can make the necessary effort for a short time. He is encouraged to do so when he is told to "stand still (or sit) for a moment" for one lateral lumbar exposure. The posture "caught" in that instant by no means characterises the motoricity of the spine. This can be verified during the neurological examination, for, if the patient, who has been trying to assume a normal attitude, is allowed to relax, he can often be seen to let his muscles go. A patient complaining of low back pain will also let his back sag, sitting as immovably as possible, preferring some backache to the distress caused by keeping the muscles constantly taut.

This view can also be defended on radiological grounds. We know of lateral lumbar radiographs of one and the same patient, on which the lordosis varied considerably, although the exposures had been taken under identical circumstances, one immediately after the other.

We would comment as follows on reduced or neutralised lordosis:

1. It may be simulated, if photographed in a chance intermediate posture between maximum flexion and hyperextension, the lumbar lordosis may seem to be neutralised.

narrowed disc arches most. The symptom is a very valuable one, because the same pathogenic significance attaches to a dorsally projecting rim – whether accompanied by a protruding disc or not – as to a hernia. Probably the prominence is brought about, partly at least, by mechanical forces (pressure on the soft vertebral body after regression of the disc in between) and partly as a reaction to pathological mobility around the disc, hence as a form of spondylosis. To our mind it is exceedingly important to diagnose posterior lipping; quite often, after the extirpation of a hernia, the projecting edge of bone runs on laterally right into the intervertebral foramen; a rut is thus formed, in which the root is compressed. Narrowing after excision of the disc (in herniatomy) may exacerbate this compression (mechanical factor). It is therefore desirable, not to say necessary, to chip off the vertebral lip right into the foramen, this either combined with foraminectomy or not (see p 607).



Indented end-plates. The end-plates are said to be indented when the bony margin of the intervertebral space does not appear on the radiograph to be perfectly rectilinear. Minor or major bulges may be associated with local clearances or sclerosis; the interpretation depends on the touchstone accepted by the examiner. The symptom has no pathognomonic value; it is merely regarded as a signal that something is going on in the disc. If the patient is

under 50, one may gather that the tissue in the disc at the site is degenerating; the same is true in a patient past 50, but the symptom loses its value as an indication of pathological change in the disc.

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We would comment as follows on reduced or neutralised lordosis:

1. It may be simulated; if photographed in a chance intermediate posture between maximum flexion and hyperextension, the lumbar lordosis may seem to be neutralised.

2. It is a defensive mechanism against pain and is maintained as long as the muscles hold out.
3. Only the reduced lumbar lordosis which does not disappear in the extreme positions (flexion — extension = functional examination) has pathological significance.

Put into different wording, this means that, even if no other cause of backache can be found, the absence of reduced lumbar lordosis should not be accepted as an indication of the "psychogenic" nature of the symptoms. Nor should a neutralised lumbar lordosis on one lateral radiograph stand for a sign of an organic lesion.

The wide (flat-oval) lumbo-sacral spinal canal. This kind of anomaly has already been discussed in the appropriate section of the literature survey (pp. (180) 159)). Widening (with erosion of the bone and expansion of the arch dorsalwards) as the result of a stenosing process, also the wide vertebral canal as a developmental anomaly at the thoracic and high lumbar levels, are known to us from the literature. Despite its comparatively high incidence, the lumbosacral wide vertebral canal as a congenital morphological anomaly appeared



This spine appears to be normal Nevertheless the canal is wider than normal; the antero-posterior diameter is short, hence the canal is oblate. Up to the present there has been no evidence that the shape of this canal is pathogenic (Woman, aged 30)

to have escaped the notice of investigators. Naturally, therefore, we cannot expect to find references to the influence of this anomaly in the production of low back pain and radicular disorders. The aspects associated with pathogenesis and therapy will be discussed in the relevant chapters. At the present juncture we have to deal with the radiological demonstrability of the anomaly and, with it, some clinical aspects of the matter.

The wide (oblate) lumbo-sacral spinal canal is a developmental anomaly. We shall disregard the canal distended by a lumbo-sacral neoplasm and the oblate one due to the invasion of osteoporotic arches.

The wide, oblate shape of the spinal canal may be the only morphological abnormality of a spinal column, but it is so often associated with signs of degeneration that we feel justified in regarding the wide (oblate) lumbo-sacral spinal canal itself as *a sign of degeneration.*

Wide (oblate) lumbo-sacral spinal canal

In neurological-neurosurgical cases	obvious	7%
	indicated	*ca.* 25%
Associated with recurrent symptoms without a true recurrent hernia		*ca.* 50%
Associated with spina bifida occulta often with other stigmata simultaneously:	transitional vertebrae	*ca.* 30%
	spondylolysis	
	sacral rachischisis	
	anomalies of the dural sac	
	anomalies of roots and ganglia.	

What is meant by "wide lumbo-sacral canal", in the sense of wider than usual, will be plain, but it is not an easy thing to describe. It is impracticable to express the morphological anomaly in exact figures or in ratios. The space can be "seen" on the semi-lateral radiograph, especially in stereo. The spinal canal is projected over the vertebral bodies on the lumbo-sacral antero-posterior plane radiograph; a visual impression of the dimensions of the spinal canal on the antero-posterior radiograph depends on the thickness, the abundance of calcium and the expansion of the pedicles of the arches. Generally, dilatation begins under the L.III arch. As the largest transverse dimensions come slightly under the L.V arch, a wide canal appears to be pyramidal. It turns out to be incorrect to express this configuration as a ratio, *viz.*, interpedicular distance L.III: interpedicular distance L.V = top : base of pyramid, because, with an extremely wide lumbo-sacral canal, the L.III arch is wider than normal, in which case the ratio would be small. The same holds for the relation between the L.IV and L.V arches.





Wide lumbo-sacral spinal canals with spina bifida occulta
These canals proved to predispose to symptoms



Wide lumbo-sacral spinal canal without spina bifida occulta This one clearly shows a pyramidal shape and proved to predispose to symptoms



Girl, aged 8
(Hemi-) transitional vertebra



Width seen in semi-lateral projection



Wide lumbo-sacral canal outlined by epidural contrast

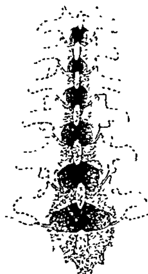
To our mind, absolute dimensions would only have some significance as comparative material with the tables of other investigators (such as Elsberg and Dyke). For, there are big and small vertebral bodies, *viz.*, small vertebral bodies with a comparatively wide spinal canal. That is why we tried to lay down a specific ratio for the respective interpedicular distances and the diameter of the vertebral bodies, but these calculations also failed to produce a specific ratio, probably because three variable factors are involved, *viz.*, 1) the size of the vertebral body, 2) the thickness of the pedicle and 3) the site of attachment of the pedicle to the body. We do not think these three factors can readily be expressed by one formula.

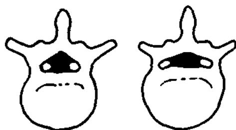
When a lumbo-sacral spinal canal appears to be wide on the antero-posterior radiograph, a better idea of the dimensions can be formed from lateral exposures:

Large dorso-ventral size and large foramina	5%
Apparently normal size	35%
Small dorso-ventral size.	60%

It is the combination of breadth in width and dorso-ventral narrowness that suggests the appellation "*flat-oval (oblate)*" spinal canal, a configuration that can be seen at operation in a large number of cases.

With this oblate shape in mind and considering that the relative width (compared to the size of the vertebral body) depends on the thickness of the pedicle and its implantation into the vertebral body, it will be evident that the shape of the spinal canal as a whole determines the shape of its lateral boundary in particular. Thus the total aspect of the shape of the spinal canal provides an impression of its lateral wall which, on account of its tridimensional, complicated structure, cannot be seen clearly in radiographs. Now that this impression has been confirmed by exploration, it proves to be of value. The lateral boundary of the spinal canal consists largely of (the wall of) the lateral recess, *i.e.*, the approach to the intervertebral foramen, an exceedingly important area where the pathogenesis of low back pain and radicular symptoms is concerned. For this, the dorso-ventral dimensions under the arch are not as important (as for the canal stenosis of Verbiest). Surprisingly and paradoxically, the diagnosis of a *wide* spinal canal tells us something about the narrowness of the area where the roots run and certain mobile parts of the spine approach each other most closely.



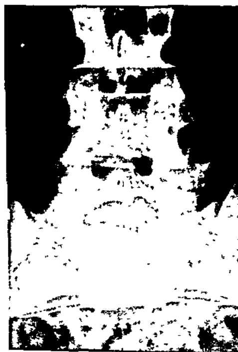


The space in the lateral recess (see pp. 147, 180) depends on the shape and expansion of the pedicles and their prolongation into the articular process or joint facets. Associated with many a malformation of the lowest part of the lumbar canal we have seen these pedicles fanning out, bending less squarely to the axis of the spinal column and overlapping; we have called them pantile extensions.

On account of its oblate shape, the wide spinal canal is also to be considered as the result of a transitional tendency. either the lumbar shape is already sacralised, or else the sacral shape has risen in a cranial direction. For this oblate shape to come into existence, the arches have necessarily to form a more acute angle with the vertebral body; the recess is then narrow and is covered exactly where the roots run by the pantile expansion of the pedicles. Moreover, the entrance to the foramina is a flatter oval. These "pantiles" can sometimes be seen radiographically; if the skeleton is rich in calcium, they cover the broad, wide spinal canal in such a way that it is scarcely visible on the antero-posterior

radiograph. With a skeleton of moderate or poor calcium content, however, thin, overlapping lamellae are scarcely visible on the picture. We have observed both conditions in which the radiographical aspect of the lamellae of the arch does not appear to tally with the findings at operation. We estimate the frequency with which the radiological examination fails to give a clear image of the spreading-out of the lamellae at roughly 25 %.

The *flat-oval* shape of the wide lumbo-sacral spinal canal has *clinical significance*, in that considerable interpeduncular distance with large dorso-ventral size is of little importance, whereas such distance with normal or small dorso-ventral size suggests a flat oval (wide) canal and, therefore, a narrow lateral recess which, to us, is a conditioning factor: the relative shortage of space predisposes to symptoms and disorders because any additional limitation of space, however trifling, may be enough to compress the roots or bring mobile parts of the vertebrae into contact with each other. In view of its frequent coincidence with signs of degeneration, we regard the wide lumbo-sacral spinal canal as itself a sign of degeneration.



Broadly, the same applies to the *asymmetrically wide spinal canal*. Its incidence is difficult to establish. The dorso-ventral size never strikes one as being enlarged on the lateral radiograph, which is not surprising since the contra-

lateral half has normal dimensions. We found that the asymmetrical shape did not absolutely determine the lateralisation of the symptoms. After all, there is no reason why a hernia should not be able to break through on the side of the canal which appears to be normal. If, however, space becomes limited in equal proportions on either side, the systems are more likely to be squeezed in the narrowed recess on the side of the laterally widened canal. When we detected an asymmetrically wide spinal canal on the antero-posterior radiograph and expressed the opinion that the patient would present symptoms on that side, our prediction proved to be correct in three out of four cases.

A wide lumbo-sacral spinal canal cannot be *diagnosed* on the basis of measurements or ratios. It depends on the subjective evaluation of a radiological aspect, as is sometimes the case in radiology (*e.g.*, the appraisal of the impressiones digitatae on the skull). Is this why a comparatively common, revealing and, in some cases, conspicuous type of constitution was overlooked?

It need hardly be said that we searched textbooks and publications for radiographs showing the wide lumbo-sacral spinal canal. The results of our quest were surprising.

Few antero-posterior radiographs are reproduced in Schmorl's textbook; some offer no opportunity for a considered judgment of the lumbo-sacral region, others show more or less clearly:

A wide lumbo-sacral canal with:

Spondylolysis	p. 39,	Fig. 63
Anomaly of spinous process	p 50	(Fig. 80) and Fig. 81
Spina bifida occulta	p 52,	Fig 83
	p. 53,	Fig. 85
Transitional vertebra	p. 58,	Fig. 92
	p. 57,	Fig. 89
	p 63,	Fig. 100
	p. 64,	Fig. 102.

While other parts of the vertebra are described amply, the wide aspect receives no comment. Many other examples could be given, but let the following suffice:

Orley, p 335, Fig 533

Von Muralt, p 105 (asymmetrically wide in association with transitional vertebra)

Bradford and Spurling, p 143, Fig 65, wide spinal canal "with false positive myelography".

Epstein,	normal spinal column,	p 58,	Fig. 31 A and C (semi-lateral exposure).
	normal spinal column,	p. 59,	Fig 32, asymmetrically wide.
	with spondylolysis,	p 350,	Fig 237 A
		p. 355,	Fig. 244 A and C (section radiograph).
	with spina bifida,	p 56,	Fig 30 (section radiograph)
	with lumbar synostosis,	p 91,	Figs 51 and 52.

There is scarcely a single book in which signs of (asymmetrically) wide lumbo-

sacral canals do not appear on the antero-posterior radiographs (Brocher, p. 38, Fig. 24; Armstrong, p. 139, Plate 16). It is a striking fact that these reproductions are mainly found in those volumes in which degenerative disorders of the back are described.

[illegible] causal relation and clinical significance of this narrowing had not yet been recognised. (Chormley, p. 175, 1934.)

Have the radiographical signs of the wide (flat oval) lumbo-sacral canal any clinical importance?

1. Faced with non-surgical syndromes, the recognition of this type of constitution helps the physician to understand vague, undefinable complaints, if he bears in mind that the wide canal is one of the manifestations of a degenerative back and in itself, therefore, accounts for the symptomatology of a weak back.
2. The diagnosis of a degenerative back calls for a more conservative attitude towards the indication for an explorative intervention.
3. With this pre-operative forewarning, the surgeon can reckon with the manifestations of this type of constitution at laminectomy and therapeutic measures can be taken accordingly.
4. On account of its frequent correlation with other pathological conditions, explorations for other anomalies have a better chance of being successful.

We wondered whether the results of this particular approach could be expressed numerically. Roughly speaking, the wide spinal canal was taken into account as a type of constitution as from 1949. This datum is interpreted in the following table in relation to approximately 1000 operations on the back before 1949 and roughly 1500 such operations between 1949 and 1st January 1954. Therefore, when comparing the evidence, the figures for the period before 1949 have to be multiplied by 3/2. The first of January 1954 was chosen as the last date to allow for the two-year follow-up period.

The investigation took place in 1957 and provides information on conditions which led to re-operation for recurrent or persistent complaints.

We have placed the patients whose first operation had taken place in the St. Ursula Clinic and those treated by other surgeons elsewhere into different groups; in the latter group the wide spinal canal was ignored, both before and after 1949.

The data thus obtained are only relatively valuable, because the numbers of cases are comparatively small after the division into two groups. Furthermore,

lateral half has normal dimensions. We found that the asymmetrical shape did not absolutely determine the lateralisation of the symptoms. After all, there is *no reason why a hernia should not be able to break through on the side of the* canal which appears to be normal. If, however, space becomes limited in equal proportions on either side, the systems are more likely to be squeezed in the narrowed recess on the side of the laterally widened canal. When we detected an asymmetrically wide spinal canal on the antero-posterior radiograph and expressed the opinion that the patient would present symptoms on that side, our prediction proved to be correct in three out of four cases.

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The noticeable frequency with which the wide canal occurs simultaneously with other abnormalities does not surprise us; least of all in this "spuriously recurrent" material. The moot point is what these figures would be – particularly the relative number of "recurrencies" after 1949 – if, owing to our special interest in this matter, a larger number of degenerative backs had not been referred to us than the average dealt with in a neurological-neurosurgical practice. It is for this reason that we cannot express an opinion on the incidence of these degenerative disorders on the basis of the numerical evidence produced by our current material.

To round off this section we present some dimensions in the table on the next page. The measurements in millimetres were made on radiographs in collaboration with the radiologist Van den Broek. They were compared with each other in various ways and some examples are given. We found no significant figures typical of the wide spinal canal. The following points may be noted:

1. There appears to be no correlation between the interpedicular distance of the L.III arch and that of L.V.
2. It is not surprising to find extremely large interpedicular distances both of L.III and L.V in spines judged by their aspect to have a wide canal. A few were also found in spines which did not give any reason for suspecting a wide canal.
3. The spinal canal may also appear to be wide in association with normal and small interpedicular distances; this is due to a certain juxtaposition of the dimensions of vertebral body and spinal canal which is untranslatable into significant figures.
4. It will be noted that the interpedicular distance of L.III is smaller on the whole than the average distance given for it by Elsberg and Dyke, *viz.*, 35 mm.

Exceedingly low values (12, 16, 17 mm) even suggest the possible existence of a relative stenosis of the spinal canal. Looking back, one begins to wonder whether the persistence or recurrence of the complaints in these cases may not have something to do with the general narrowness of the canal. (All these particulars were obtained on radiographs of patients who underwent reoperation.)

5. In our material the extremely large interpedicular distance of L.V was liable to be far beyond Elsberg and Dyke's upper limit of 39 mm, *viz.*, 40, 42, 43, 44, 45 mm.
6. Roughly speaking, the greatest disparities in size of vertebral body and in interpedicular distance (L.V in relation to L.III) occur in association with the wide spinal canal with wide interpedicular distance of L.V.

only one aspect of the modified technique is being considered but, assuming that it also evolved elsewhere, the comparison with patients operated upon for the first time elsewhere is apt.

	First operation	*Hernia at re-operation*		*No hernia at re-operation*	
Before 1949	Ursula		50		23
	Elsewhere	18		8	
After 1949	Ursula		25		20
	Elsewhere	25		14	
		43	75	22	43
		118		65	

Re-operation at which a hernia was not found

	First operation		*Normal spine*	*Signs of degeneration*	*Signs of degeneration + wide canal*	*Wide canal only*	*Total of wide canals*
Before 1949	Ursula	23	4	4	7	8	15
	Elsewhere	8	2	3	1	2	3
After 1949	Ursula	20	4	7	8	1	9
	Elsewhere	14	3	1	9	1	10
					25	12	37

It will be seen that in 37 of the 65 re-operations (recurrency without hernia) the spinal canal was wide. This is *ca.* 50%. In neurological-neurosurgical patient material it is *ca.* 25%, in mixed material (neurology, internal medicine, gynaecology) *ca.* 10%. Another striking fact is that in the Ursula Clinic the number of spurious hernia "recurrencies" diminished in an absolute sense after 1949 and still more so relatively to the total number of operations, *viz.*, $23 \times 3/2 : 20 = 34\frac{1}{2}$ 20

Reviewing the radiographs of gynaecological cases, we noticed to our astonishment that nearly all cases of carcinoma uteri in this series had a flat wide spinal canal (!). Though we mention this only in passing and it is by no means our intention to suggest that this coincidence is anything but mere chance, we do intend to investigate this matter more closely.

35	35	35	36	36	37	37	37	37	38	38	39	40	40	40	40	42	43	44	45
60	70	55	75	58	64	63	63	70	66	70	66	65	88	73	70	65	70	66	80
35	28	20	26	27	28	25	30	19	38	26	28	23	40	32	31	22	30	38	39
55	55	47	58	50	47	54	56	45	60	60	54	54	71	59	59	52	56	55	67
25	35	20	39	22	27	26	26	33	28	32	27	25	48	33	30	23	27	22	35
20	27	27	32	23	19	29	26	26	22	34	26	31	31	27	28	30	26	17	28
0	7	5	10	9	9	12	7	18	0	12	11	17	0	8	9	20	13	16	6
5	15	8	17	8	17	9	7	25	6	10	12	11	17	14	11	13	14	11	13

32	32	32	32	34	36	36	36	37	38	39
61	67	70	60	68	54	70	56	64	60	62
30	20	20	32	27	30	27	28	29	28	32
57	56	60	49	59	48	50	49	53	54	62
29	35	38	28	34	18	34	20	27	22	23
37	36	40	17	32	18	23	21	24	26	30
2	12	12	0	17	6	9	8	8	10	7
4	11	10	11	9	6	20	7	11	6	0

34	37	37	38	40	40
59	62	60	63	60	80
20	36	12	18	30	42
50	56	50	54	50	70
24	25	23	25	20	40
30	20	38	36	20	26
14	1	25	20	10	2
9	6	10	9	10	10

Measurements in Millimetres on Radiographs

Wide lumbo-sacral canal				
Interpedicular distance	L.V	29	32	34
Width vertebral body		64	58	60
	L.III	27	27	30
		50	50	54
Width vertebral body - Interpedicular distance	L.V	35	26	26
	L.III	23	23	24
Difference between interpedicular distances of L V and L.III		2	5	4
Difference between widths of vertebral bodies L.V and L.III		14	8	6
Spine with signs of degeneration though canal not wide				
Interpedicular distance	L.V	28	30	32
Width vertebral body		70	70	70
	L.III	17	17	16
		54	56	60
Width vertebral body - Interpedicular distance	L.V	42	40	38
	L.III	37	39	44
Difference of interpedicular distances of L V and L III		11	13	16
Difference in width of vertebral bodies L V and L.III		16	14	10
Neither signs of degeneration, nor wide canal				
Interpedicular distance	L.V	26	32	33
Width vertebral body		52	62	50
	L.III	12	26	26
		45	52	43
Width vertebral body - Interpedicular distance	L.V	26	30	23
	L.III	33	26	17
Difference between interpedicular distances of L V and L III		14	6	7
Difference in widths of vertebral bodies L V and L.III		7	10	7

Hernia verified at operation was found to be accompanied by radiographically detectable intervertebral narrowing in 31.2% cases according to Armstrong (p. 99) and in 26% according to Reischauer (p. 37).

Disc narrowing L.IV – L.V with hernia (at the same level)			*Disc narrowing on radiographs*	
Norlén	68%		Krayenbühl	*ca.* 50%
Waris	77%	(p. 59)	Love	40%
			Norlén	35%
			Waris	30% (p. 58)

The combination of hernia and disc narrowing and local secondary spondylarthrosis occurs more frequently, as pointed out by Brocher (p. 30) and Bradford and Spurling (p. 118). Krayenbuhl (p. 39) reports this combination in 68%, adding that the hernia was situated at the level of the narrowing in half the cases, at a different level in a quarter of them and that in the remaining quarter more than one intervertebral narrowing was found, though only one hernia. Williams and Yglesias (Bradford and Spurling, p. 118) found a lesion of the lumbo-sacral disc in 74 per cent. of cases of "sciatica" due to pathological bone or joint changes.

In point of fact, it remains very much to be seen whether narrowing of the lumbo-sacral intervertebral disc may be compared as a symptom with the narrowing of the L.IV – L.V disc. It was our impression that the narrowing of the L.IV – L.V pointed more significantly to an accompanying hernia than the lumbo-sacral narrowing, taking into consideration the fact that various factors may be responsible for the latter. A comparative investigation produced evidence in support of this conclusion; also, the figures found agree very well with those given in the literature just referred to.

	Disc narrowing
300 back radiographs (neurological and internal indication)	14%
300 "sciatica" not verified at operation	32%
300 verified hernias (IV–V and V–I)	52%

We were able to subdivide this latter group of 300 hernias as follows:

Disc narrowing		*Hernia*	
24%	IV–V	IV–V	37% correct
11%	V–I	V–I	
2%	IV–V + V–I	IV–V + V–I	

As against this, these last-mentioned disparities in association with the apparently normal canal are conspicuously at their greatest in the cases of small interpedicular distances of the L.V arch. These differences depend on the size of the vertebra as appearing on the radiograph, with projection errors and individual variations included. Subsequent measurements made have shown, moreover, that the "wide canal" diagnosis arrived at by subjective assessment *of the total aspect of the spine is not demonstrably adherent to any particular* size of the vertebra. It is not premature to conclude, therefore, that the eye was not misled in this subjective evaluation by the dimensions of the vertebra. Furthermore, considering that the dorso-ventral dimensions on the lateral projection only failed to bear out the surmised broad, flat oval shape of the canal in 5 % (pointing out at the same time that this shape, suspected pre-operatively on the basis of the aspect on the radiographs, was seen to be so at operation in the vast majority of cases), we believe that the all-round aspect of the antero-posterior plane radiograph provides a reasonably reliable impression of the width of the spinal canal.

Naturally, wide arches will also often go hand in hand with a wide canal. In our table there are no extremely small interpedicular distances together with what seem to be wide canals. All the same, the width of the arch, or the interpedicular distance, is not necessarily a determinant for the width of the whole canal. The canal is wider in between the arches than under them. It is precisely between the arches that the wall of the canal is formed by the lateral recess, just in front of the foramina where the roots run. Owing to softness of contours, masking by the roof-like spread of the arches and differences in projection, we have not yet succeeded in determining the width of the lateral recess by exact measurement on radiographs.

Narrowing of the disc. Greater value was formerly attached to this symptom for the diagnosis of "hernia" (Glorieux) than at the present time. Opinions differ, varying from "of little value, valueless" to "liable to give rise to misunderstanding and faulty diagnoses".

Epstein (p. 56). "It is the author's observation that narrowing of the lumbosacral interspace in itself is not of any clear-cut diagnostic significance".

Reischauer (paper read to the Soc of Bavar. Surg., July 1956): "Nothing is more misleading (for level diagnosis) than the radiological findings".

Lewin (p. 90) "Normal X-rays do not mean the back is normal. A man's X-rays may be absolutely normal, still he may have a very painful back. Another man's X-rays may show considerable changes, but he may have no back complaints at the time. The X-ray usually reveals old troubles. The symptoms and special tests disclose the present trouble".

6. The disc under a transitional vertebra is always narrow.
7. The L.V – S I disc may be congenitally narrow (Willis, see Bradford and Spurling). To our mind, manifestations of transitional tendencies are then so often found, that congenital narrow L.V – S.I discs are themselves a sign of a transitional spine.
8. Residual conditions after a hernia operation.
9. Residual conditions after a "cured" disc lesion.
10. Residual conditions after a "cured" infectious process.



"Pantile" fanning out of S.1 (and L.V) arches



Exceedingly long sacrum



Rudimentary S.I – S.II disc

9%	IV–V	V–I	15% wrong
1%	V/I	IV–V	
1%	V–I	IV–V + V–I	
4%	IV–V	IV–V + V–I	

(We did not group by other standards (*e.g.*, age in view of possible physiological disc narrowing), because our only concern was the correlation between intervertebral narrowing and hernia at the same level.)

Hence in one to every three or four disc narrowings (15 to 52) the hernia was not situated at a corresponding level. This grouping also shows that hernia occurred without intervertebral narrowing in 48%. The L.IV – L.V disc was found to have narrowed in 37% and the L.V – S.I in 15%.

The decision as to whether a disc has narrowed or not is influenced to some extent by the examiner's own criteria. Comparison with the thickness of discs at other levels offers little corroborative evidence; other discs may also have narrowed or may even have thickened. The subjectivity of the evaluation becomes apparent when radiographs are studied by more than one person. To test the degree of our own subjectivity, we examined other groups. Although the figures are different, roughly speaking they were in agreement with those of the preceding enquiry.

100 selected IV–V hernias were accompanied in 31% with IV–V disc narrowing;
100 selected V–I hernias were accompanied in 22% with V–I disc narrowing.

Perhaps the IV–V disc is too often said to be narrowed and other factors are too often held to be responsible for the aspect of a possibly narrowed V–I disc.

Disc narrowing is by no means pathognomonic for the clinical picture of hernia nuclei pulposi. *E.g.:*

70 "negative explorations" in the period before degenerative backs were diagnosed. 13 disc narrowings = 18%.

50 negative explorations for hernia in so-called degenerative backs: 14 disc narrowings = 28%.

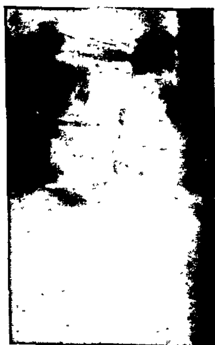
What are the factors that can cause intervertebral narrowing or its simulation?

1. Hernia nuclei pulposi
2. A disc lesion.
3. Age. Past middle age the intervertebral discs narrow physiologically.
4. Faulty projection. Narrowing is difficult to judge on the antero-posterior plane radiograph
5. Wrong interpretation of level on the lateral lumbar plane radiographs. It is sometimes difficult to determine the correct level on the lateral radiograph, especially if there are transitional vertebrae; *e.g.*, a rudimentary S.I – S.II disc may be regarded as a narrowed L.V – S.I disc.

Examination of function

For this a number of lateral radiographs are made, with the patient, either sitting or standing, bent forwards and backwards in full flexion; the sitting posture is preferable, because the patient is more relaxed. An impression is received in this way of the function of the spine. More especially, changes in the lumbar lordosis become visible, while (subclinical) displacements can be seen which were overlooked or did not appear at all on one lateral exposure. Mindful of the mechanism inducing functional listhesis, it will be clear that the tendency to glide may be masked by a rigid locking of the lumbar spine in a middle position on an instantaneous photograph, on which the listhesis is then invisible.

The value of this examination is recognised everywhere. Roughly speaking, one in every twenty patients seems to call for examination of function. In half the cases in which this indication derived from pathological motoricity suggesting displacement ("working" the spine up on rising), a functional





Synostosis – posterior lipping.
Functional listhesis appears clearly on hyperextension

Manifestations of transitional vertebrae. In the foregoing pages we have several times dwelt with emphasis on any kind of sign which could be taken as a manifestation of a transitional tendency. We think it would be useful to recapitulate these signs.

1. The merging of the 24th vertebra into the sacrum (sacralisation).
2. The 25th vertebra standing detached above the sacrum (lumbarisation).
3. There are 6 "lumbar vertebrae" owing to lumbarisation of Th.12.
4. Rudimentary ribs on L.I.
5. Uni- or bilaterally enlarged lateral process of L.V, either or not merging into the sacrum and pelvis.
6. Sacral shape of the bony spinal canal at L.V – S.I.
7. "Pantile" fanning out of the S.I (and L.V) arches (see radiograph p. 463).
8. Pseudo-spina bifida (bony defect between S.I and S.II).
9. Exceedingly long sacrum (see p. 463).
10. Extension of the septa between the intervertebral canal of S.I and S.2 roots in a proximal direction (see p. 603).
11. Rudimentary S.I – S.II disc (see p. 463).
12. Congenitally narrowed L.V – S.I disc.

With the interpretation of all these indications, transitional tendencies prove to be demonstrable in roughly 35%.

The list could be amplified with minor changes to arches or spinous processes.

Manifest sacralisation or lumbarisation was found to occur in about 7%.

Upon certain indications, the radiographical examination is elaborated, with what frequency it is difficult to say owing to periodically changing standards of indication. In approximately 15% the radiographic examination was supplemented with the *semi-lateral* lumbar plane radiograph. The indication depended chiefly on the detection of retroposition on the lateral radiograph, which suggested the existence of spondylolysis. The examination of nearly one in every four cases X-rayed for these reasons was rewarded by the disclosure of an abnormality in one or other arch, when it became possible to classify the listhesis as a true spondylolisthesis. The detached arch, without displacement, is rare; its probable existence was demonstrable on the semi-lateral radiograph Further, oblique exposures, especially if they are stereoscopic, are instructive on the width of the lumbo-sacral canal, which then looks like a bag-shaped space of amazing dimensions. These dimensions are all the more astounding if, through lack of experience or misled by the apparently normal structure of the spine projected over the canal, the examiner received no such impression from the antero-posterior radiograph.

of the author, it would be rash to conclude that there are no radiographically demonstrable abnormalities without an examination of function. We shall not enter further into the importance of this examination, because the demonstration of abnormal changes is a radiographic technicality.

Exposures in special projection

In exceptional cases the need may be felt for further radiological information on the condition of the spine, an example being the *lateral detail* projection of the lumbo-sacral region, when the corpulence of the patient or the over-projection of the pelvis impedes the observation of details on the ordinary lateral radiographs. One drawback is the larger amount of rays involved in fluoroscopic screening.

Faced with unclear syndromes of the S.1 and S.2 roots, a clinician may wish to know more about the position of the *sacral foramina* so that he may trace the probable course of these roots. Likewise, ambiguous syndromes associated with transitional vertebrae may make it necessary to know more about the correct appellation for the transitional tendency noted. We have expatiated in the foregoing on the significance in neurological diagnosis of the correlation between the anatomical name given to, and the clinical function of, a root. It is scarcely ever possible to tell whether there is a transitional tendency in a cranial direction or in a caudal one merely by the aspect at the lumbo-sacral level. The level of the 24th vertebra can be established with high probability by counting out on the *radiographs of the whole spinal column*. It may even be necessary to look out for the possible assimilation of vertebra C.I and, to make sure, have a lateral radiograph of the skull made in strong flexion.

We should also like to point out at this juncture that it may be desirable to have *post-operative check-up radiographs* made. Inconclusive level determination at exploration and unsatisfactory findings respecting myelographic recesses in the contrast medium, thought to be convincingly pathological, call for a greater measure of certainty before the patient is discharged. A useful mark in such cases is a silver clip left behind at operation. We used to introduce silver threads next to the root concerned if any uncertainty remained as to the nomenclature of the roots; this, in itself, may perhaps be unimportant therapeutically, but is an integrating link in obscure syndromes for the prognosis and indication for another intervention if the complaints persist. When the course of the roots had been clearly established in this way on the check-up radiographs, the threads were removed simply by traction of the thread-ends projecting from the closed wound (See radiographs next page).

When back disorders persist, the physician's advice is often sought on the

listhesis was observed which had previously been invisible on the single lateral radiograph. Sometimes, too, this examination takes the form of a series of antero-posterior exposures for the purpose of establishing the nature of a scoliosis (Hadley, see Weersma). It is said to reveal whether a scoliosis proceeds from defects in the spine or is caused by painful muscle tension as the result, possibly, of pathological conditions in other organs. An impression is gained of the axis of movement between individual vertebrae by this same examination. Wiles (see Armstrong, p. 28) suggested that this axis is not fixed, but changes during movement. We observed no relative displacements between the vertebral bodies in physiologically functioning spines. It would be more cautious, and perhaps truer, to say that, if any displacement is observed, we regard the mobility of the spine as not being physiological.

The examination of spinal function is very important. Quite unexpectedly, it reveals abnormalities like displacements, asymmetrical narrowing of the disc and other changes pointing to a pathological condition. In the personal opinion

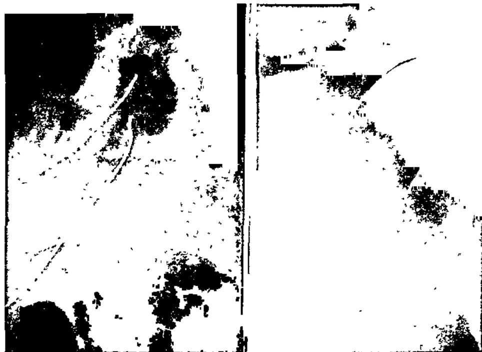


Asthenia *sive* dystrophia musculorum progressiva. Functional listhesis appears clearly on full flexion

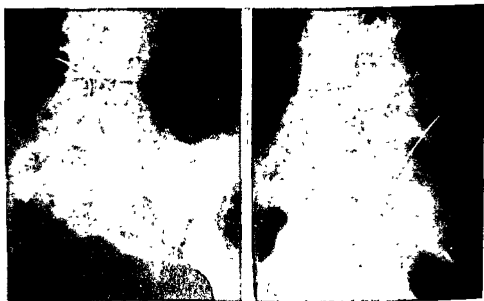
questions of resumption of work and the patient's future mode of life. In doubtful cases it may seem proper to adduce the evidence of radiographs in support of the advice given. This should not be resorted to merely to charge the pronouncement with greater suggestive force. It is a great responsibility to accuse a patient of exaggeration, simulation or psychogenic reactions in any other sense and then to encourage him to take up work. In such cases we like to make sure, radiographically, that there is no vertebral retroposition. Cases are known in which the organic basis of residual back complaints was unexpectedly disclosed.

The value of a diagnostic examination depends on how it influences the diagnosis made and the degree in which the stated indication and the procedure during the surgical intervention itself are directed by it. Hence it is for the diagnostician and the therapeutist to judge of the value of the radiological examination or its usefulness to the patient.

It has already been stated that a simple antero-posterior and lateral radiological routine examination provides merely a rough, general impression; indeed, one would not even expect to see several important details on these radiographs. To gain the fullest possible information, with some assurance that probably no important data have escaped attention, the patient would have to submit to something between nine and twelve exposures (leaving special methods of examination, like myelography, out of account). For, by omitting some particular section of the examination, the practitioner is left in doubt as to whether the abnormality which that particular examination would have been performed to detect does or does not exist. The relative value of the routine examination has, therefore, to be weighed against the risks attaching to an extension of the examination. These risks are by no means illusory, as radiologists have lately been at pains to point out. The American Journal of Roentgenology (1957, p. 961) states that the dosage of R upon the skin for a lumbar-sacral exposure (on account of the necessary tension, time and size of field) is more than five times that needed for a radiograph of the skull. The number of scattered rays reaching the gonads (especially the testicles) is about 60 times greater for a lumbo-sacral X-ray than for a cervical radiograph. The dose of rays for one minute's lung fluoroscopy is approximately comparable to that needed for 200 lung radiographs! Without entering into exact physics, it is a matter of fairly simple calculation that the number of scattered rays on the testes after lumbar-sacral fluoroscopy lasting one minute (as in myelography, for instance) is $200 \times 60 \times 1$ to $5 => 12{,}000$ times as great as that resulting from one skull exposure. Even if these figures are disregarded, it can be stated



Note the threads along the nerve roots S.1 in the lateral projections!!



Note the threads along the nerve roots S 1 in the lateral projections!!

without a special indication. We might perhaps estimate the frequency at approximately 10%.

(b) How important is the information thus obtained? This depends partly on the nature of the disorder, partly on the attitude of the diagnostician and therapeutist. Plates which are apparently just tucked away in a file without being studied by the doctor who is treating the patient can scarcely be said to have much value. Useful as may be the expert description by the radiologist, there are still too many details which have to be *seen* to be understood. Therefore, the diagnosing practitioner must be able to read radiographs covering his own terrain.

(c) To whom are these special details of *importance*? If, in any event, the practitioner only performs routine operations (*e.g.*, herniatomy or spondylodesis), it is to little purpose to prime him with details which he does not use. If it be said that such an allegation is premature, we should counter that the very expression "negative exploration", by which is meant "no hernia", proves the object for which the laminectomy is then undertaken. The number of cases in which "low back pain", and not some other abnormality of the spine, has been treated by spondylodesis is legion. Detailed information on the condition of the spine is useful only if the practitioner is in the habit of availing himself of it to good purpose.

(d) A simple X-ray examination has become so commonplace that it need not affect the patient *psychically*. On the contrary, if he is told that the radiographs show up nothing serious, it will reassure him considerably. There is no reason at all why the patient should be told more than is good for him; but, if he is asked to come back for a more thorough examination, a psychotrauma may well ensue. Once he has become imbued with the fear of suffering from some grave disease, it is difficult to talk him out of it, especially if his fears are sustained by persistence of the trouble in his back, for which the X-ray examination was undertaken in the first place.

(e) Sometimes a patient himself asks to be radiologically examined, whereas the medical grounds for such an examination seem to be dubious. It is of no avail to hint at the *expense*; the patient is in no position to judge either of the value or the necessity of the step; he insists, if only from fear of leaving something undone to restore or maintain his health.

In an extensive radiological examination the body is exposed to four or five times the amount of radiation that would be involved in routine antero-posterior-lateral films. Are risks incurred and is it right, therefore, to proceed with it as a routine measure without special indications? The desirability or admissibility of this radiological examination stands or falls with the answer to

confidently that the danger of radiation in fluoroscopy far and away exceeds that entailed in a radiograph. This applies in no less measure to direct radiation of the gonads, especially the ovaries, perhaps, which lie more directly within the field of radiation and cannot easely be protected.

Realising all this, we find ourselves confronted with some contradictory problems, *viz.*,

Few surgeons, if any, will be prepared to undertake a laminectomy without some radiological evidence respecting the condition of the spine.

Without such evidence, no diagnosis would be considered as complete.

Yet, in about 85 % of cases many seem to be satisfied with a simple antero-posterior-lateral routine examination.

Yet, this routine examination provides only roughly approximative information and thus many important abnormalities must of necessity fail to be seen.

It might therefore be said that radiological examination of the lumbo-sacral spine as adopted in practice (85 %) has only fictitious value; or, to look at it from another angle: The diagnostician, not realising how little antero-posterior-lateral radiographs are capable of revealing, derives reassurance from their presence. Now furthermore considering that the interpretation of the abnormalities which *are* visible in these radiographs is governed by subjective criteria and that an appreciable percentage of these abnormalities (in disc narrowing about 29 %) misleads us, we shall the better understand the statements made in the literature on the value of X-ray routine examination (Epstein, Lewin, Reischauer). (Lenhard, p. 428: "Roentgenographic interpretations are not reliable for accurate localisation of the lesion".)

We may add as a personal note that at first the fulminating, condemnatory judgment in Reischauer's writings seemed incomprehensible to us, or at least highly exaggerated; *but it was his opinion that led us to embark upon this analysis*, which resulted in the conclusions set forth above.

The next obvious deliberation is whether this implies that a comprehensive radiological examination is indicated for every patient. The decision depends on the following factors

(a) In that case, *how often* would important information be obtained which is not furnished by the routine antero-posterior-lateral examination? It is impossible to establish this frequency. When, in the past, the examination was extended, there was some special reason for it; hence the frequency with which abnormalities were then found (*e g*, lysis of the arch on the semi-lateral: about 25 %) was flattered An exact figure cannot be given, for the simple reason that a complete examination (roughly 9 to 10 radiographs) was not undertaken

pression that this dilemma is not well thought out. There is numerical evidence showing that medical radiology contributes no mean percentage of humanly dangerous radiation, including, as far as these are known, the results of nuclear fission processes. (A person of 35 is said already to have received radiation of 5 R through cosmic-telluric influences.)

Back disorders are endemic and their treatment, through a variety of circumstances, is claiming more and more of our attention. If radiation is a real danger to humanity, it is incumbent upon us to devise precautions and enforce them in all fields of diagnostic medicine. We should even consider whether the taking of diagnostic risks is not balanced by the reduced dangers through radiation thereby achieved. Lumbo-sacral radiological examination is implicated because it is exceedingly dangerous and because a comparatively large proportion of the population is liable to be exposed to it.

Suggested points to be considered before prescribing lumbar-sacral radiological examination

1. Never prescribe by request unless it is medically indicated.
2. Routine antero-posterior-lateral pictures to be taken if indicated by evidence of preceding (neurological-orthopaedic, etc.) examination, provided such evidence also holds out promise of benefit from treatment which can only be carried out on the basis of the radiographic data obtained.
3. Extensive explorative examination only to be carried out if indicated by preliminary examination and the routine antero-posterior-lateral radiographs.
4. If the preliminary examination requires that a radiological function examination should be made, the lateral routine plane film becomes superfluous.
5. Radiographs in special projections should only be made if specifically indicated for therapeutic measures.
6. A particularly strict indication should be demanded for exposures requiring screening; but fluoroscopy should be avoided as far as possible when making certain pictures.

Miscellaneous. The first examination ought to be successful. Hence the importance of referring the patient from the outset to a radiological department having experience in the examination required.

Every X-ray department should satisfy the highest standards in point of equipment, personnel and administration, to avoid unnecessary repetition and exposure of the staff unnecessarily to the dangers of radiation.

Radiological Examination with Contrast Media

Myelography. Every contrast medium disturbs the composition of the cerebro-spinal fluid Depending on the irritation set up by a particular contrast medium

these questions, compared with which all the preceding objections are merely of relative importance. Owing to the very many imponderables, the answer cannot be given in precise terms.

On the radiological side, the following possibilities, here roughly outlined, are taken into account.

(f) With present-day technique and precautionary measures, the *danger* to the patient of *exposure of the skin to X-rays* for diagnosis is trifling. Yet it was pointed out at the conference of German neurosurgeons on glioma (Bonn, February 1958) that wounds do not heal normally after many pictures of the skull have been taken.

(g) The consequences of radiation or ray scattering to the gonads have been the subject of many recent studies. Mutations may result from the exposure of germ cells to X-rays. The lethal and deforming effect of radiation upon the young foetus is known; its deleterious influence on immature sex cells, long before they are fertilised or fertilise, is feared. Little doubt is any longer entertained that these direful effects are liable to come out in future generations.

Now, in lumbo-sacral radiological examination, a large quantity of rays reach the gonads. The critical limit beyond which the dose becomes injurious cannot be even approximately estimated; it might be comparatively low if precisely that germ cell leads to progeniture which is struck by radiation – perhaps of only slight intensity – in a certain sensitive stage of the mitotic process. Although the deleterious effect of radiation upon the gonads need not be determined quantitatively in an absolute sense, the probability of injurious consequences *is* quantitatively progressive. Therefore, seeing that the consequences are imponderable, the indication for an examination of the kind should be bound to strict necessity or intrinsic utility.

Above a certain age (45 in women?) the chances of progeniture may be esteemed so small that the above objection would cease to exist.

(h) We do not yet know whether the *damaging effect upon the haemapoietic organs* would increase considerably beyond the age of 45, *viz.*, in a not absolutely quantitatively progressive correlation. The practitioner does, however, bear in mind that the bone marrow above this age may be disorganised in its activity by even a small dose of rays. Young people, too, sometimes fall victim to leukaemia, aplastic anaemia and so forth.

Weighing the usefulness of this radiological examination of the lumbo-sacral spine to diagnostician and medical practitioner against the drawbacks (especially the dangers of radiation which the radiologist suspects), we are all too often in a quandary when faced with the necessity of formulating a well-founded indication for this examination It is, moreover, our personal im-

become known until about ten years after. Admittedly, oil myelography produces sharp contrast, but this is only a relative advantage, since many faulty myelographic diagnoses are due to "seeing too much" (positive in negative finding). Owing to the viscosity of the oil, numerous artificial recesses may be induced (as may be seen while screening) by letting the oil ascend or descend slowly.

It became clear to us that the inconclusiveness of indeterminate contours in the air myelogram is more useful than the sharply outlined configurations of the myelogram with oil contrast medium, in which, however, those of pathological significance have to be distinguished from the non-pathological ones. This subjective evaluation is such a pitfall, that, in the ultimate, the concrete diagnostic and localising value of the oil-myelogram proves to be no greater than that of the air-myelogram.

(d) In large measure the *aqueous contrast media* offer the same relative advantage. They are, however, so painful to the patient that lumbar anaesthesia or total narcosis is required. Furthermore, there is the very real danger of these strong irritants reaching the thoracic marrow or the base of the skull if the posture is wrong, sometimes with lethal consequences. The necessary anaesthesia, indispensable strict precautions and duration of this method are points against its adoption for routine purposes.

After this ample discussion, we wish to make it plain that we do not intend to enter into the technical details of myelography. If any one of these methods offered outstanding advantages, medical practice would have resorted to it long ago. Our aim is to appraise the value of the examination in the diagnostic and indirect therapeutic sense, and to weigh the drawbacks, trouble and time needed against the importance of the information obtained. Does the patient benefit if the practitioner asks the diagnostician to have the diagnosis he has made on the basis of neurological symptoms confirmed by myelographic data, irrespective of the method by which the most reliable data are obtained? In other words, has it been necessary in the past to complete the neurological diagnosis with myelographic particulars and has this diagnostic intervention (by whatever method carried out) answered its purpose? The aetiological and localising diagnosis (with respect to hernia) is simultaneously approximated by a myelographic examination; so we may ask again whether the information thus received is of such use to the practitioner – and therefore to the patient – as to warrant the procedure?

We here leave out of consideration any differences there may be between suboccipital and lumbar myelography; also the combination of myelography and tomography, as we are concerned only with the question as to whether an

and the sensitivity or reaction of the intradural tissues, it takes a month or longer for the fluid to be cleared. Cases are known in which the composition of the fluid was disturbed for many years and it was then said to be due to arachnoiditis. As few back patients underwent a second lumbar puncture, this was not verified statistically; data on fluid composition in cases of recurrent complaints are not revealing, as it is precisely then that a persistent or new disorder is assumed to exist which might itself induce changes in the fluid.

The following results of an examination, undertaken despite the negative tenor of the answers to the pertinent questions, may clarify some comments to follow. After the introduction of *lipiodol*, this contrast oil was found to remain suspended in droplets for varying lengths of time (caviare-lipiodol). The question to be settled was whether this had anything to do with the composition of the fluid, more particularly with its protein content.

In 71 lipiodol myelographies:	31 times droplet formation	(= 43%)
In 54 of these, fluid composition known:	23 times droplet formation	(= 43%)
In these 23 cases with droplet formation we found:	8 times protein content	$>$ 0.035‰
	3 times protein content	$<$ 0.021‰
In the remaining cases without droplet formation (54–23)	6 times protein content	$>$ 0.035‰

As the protein content is raised roughly by $>$ 0.035‰ in about 24% to 31% of back patients (hernia), it was to be inferred from the results that there was no correlation between protein elevation and the formation of lipiodol droplets. What did emerge from this investigation was that droplets persisted longer in fluids which already contained $>$ 0.035‰ protein at the time the lipiodol was introduced. Since the introduction of oil is known to raise the protein content, it may be that the persistence of droplets is due to increase in protein after the introduction of the oil.

The following should be considered when assessing the profitability of myelographic examination to the patient.

(*a*) *The method* (gases, air, re-absorbable oils, acqueous solutions as contrast medium).

We shall not discuss the use of *lipiodol*, because its dangers have been sufficiently stressed in the literature (Prick and Hoefnagels and others) and because this medium is no longer employed.

(*b*) *Air myelography* is said to be the least injurious, but it is also alleged to produce less reliable indications; moreover, it causes the patient considerable distress (headaches, pre-shock, prolonged after-treatment 4 to 7 days).

(*c*) The *re-absorbability* of the so-called re-absorbable oils used is relative; some patients continue to carry remains of them in their dural canal for years (Zukschwerdt, p. 114). Although the injurious effect upon the intradural tissues does not appear to be very serious, the fact should not be overlooked that the bad effects of the regular use of lipiodol (arachnitis, etc.) did not

H.N.P.	Fluoroscopy right	Fluoroscopy wrong	Myelographs right	Myelographs wrong	Recesses on pictures tally right	Recesses on pictures tally wrong
V – I	26+	– 17	35+	– 23	19+	– 2
IV – V	37+	– 8	54+	– 11	17+	– 1
III – IV	1+	– 6	1+	– 4		
	64+	– 31	90+	– 38	36+	– 3
	95		128		39	

In 95 of the 128 cases a myelographic diagnosis was given with fluoroscopy. In 39 cases there were enough films to compare recesses; by holding them together, one behind the other, against strong light, it was possible to see that certain contours consistently covered each other, even when these contours did not form part of a complete recess. This *consistent tally* was found to provide clear evidence of an abnormality at that level. In this series the many faulty diagnoses were in most cases due to an assessment of antero-posterior myelograms.



After 1949, our myelographic diagnoses with the help of air or ethiodan examination, on antero-posterior and lateral films, combined with the findings at fluoroscopy, proved to be wrong in 30% to 20% within the above terms. In selected cases, the clinical picture having already decided diagnosis and localisation, we were able to reduce this percentage of errors to 10% (when either the localisation only was at fault or else that and the diagnosis). The findings, subdivided, were as follows:

Ursula Clinic	*Myelography* correct	wrong
Back operations (including negative explorations)	70%	30%
Backs with signs of degeneration	68%	32%
Backs without signs of degeneration	80%	20%
First myelography for patients whose symptoms persisted	71%	29%
Of these, genuinely recurrent (hernia at second operation)	74%	26%
"Recurrency" in these without hernia at second operation	69%	31%
Ditto with signs of degeneration	62%	38%
Without signs of degeneration	79%	21%

As the group of "back operations" consisted of 500 cases, the records give a reasonably reliable impression, *i.e.*, 70 +, 30–. Subdivision led to such small groups, that the same cannot be said of the evidence provided by them. For all that, it would seem to emerge that myelography is least reliable for backs

abnormality is or is not reliably demonstrable. For the same reason, the nature of the abnormality is also of secondary importance in this context.

Selection. In some clinics, myelography is part and parcel of the routine examination for the diagnosis of back disorders; in others it is only resorted to as a complementary measure, or in an attempt to locate the exact position of an abnormality already diagnosed, but only in cases indicated for surgery. In between these two extremes, it seems that elsewhere the indication for myelography is made to depend on circumstances, *e.g.*, if there is any doubt about the diagnosis. This selection will no doubt influence the percentages in which the myelographic findings pointed to the probable existence of an abnormality, and those in which this assumption later proved to be correct. It is clear, for instance, that there is less likelihood of disclosing a cauda equina tumour and more likelihood of making a wrong diagnosis by routine myelographies than if myelographic examination for a cauda tumour is only undertaken in a given case after the clinical picture (showing, among other things, a conspicuously high protein content of the fluid) had suggested it.

Subjectivity. As evaluation by team-work has proved, the interpretation, not only of the findings on screening, but also of the exact black-and-white reproduction on the pictures, is coloured by the subjectivity of the examiners.

We do not consider it to be within our terms of reference to weigh all these fine points one against the other. It is even more impracticable in any comparison between our data and those obtained in the literature, in that the circumstances under which the examinations referred to in the latter took place are not generally stated.

The important point to us is to find legitimate grounds for resorting to myelography and, with this purpose in mind, we thought clearer insight would be obtained by building up a synthesis than by digressing into minutiae with reference to our own records.

The value of myelographic examination depends upon the information it provides. If it is correct, disclosing a hernia, for example, at the level predicted, we say that the myelography is right, but wrong if the prediction is not corroborated later at operation, as when there is no hernia or some other lesion exists at an unsuspected site not hinted at by the myelography.

Between 1934 and 1949, 128 hernia cases verified at operation were examined with the help of lipiodol myelographies, sufficient data turning out to be known. The following chart does not, therefore, include wrong myelographic diagnoses due to the prediction of a hernia, whereas the subsequent exploration for hernia was negative. (The data have been verified in collaboration with the radiologist Van den Broek.)

O'Connell (p. 767, see Lewin): Myelography and routine lumbar puncture unnecessary.

Reischauer (p. 38): "...unzuverlässig, schädlich, überflüssig".

Scoville *et al.*: "...clinical evidence of ruptured disc is more important than the myelographic evidence..." (Spiegel 1957, p. 309).

Spurling (p. 73): "...limited to special indications".

Ståhl: "Negative myelograms and the absence of neurological signs by no means exclude the possibility that the sciatic syndrome is due to a herniation".

Thurel (p. 26): Myelography not always necessary.

Voris (p. 122): In cases without objective neurologic findings.

It can be inferred from these surveys that myelography furnishes reliable information in roughly 70% to 80% and is, therefore, at fault in 20% to 30%. Several very high percentages were found to have been calculated on the basis of comparatively small groups (50 to 70 cases). This suggests a selection, for instance the restriction, already referred to, of indicated myelography to classically neurologically clear cases, in which myelography was only expected to provide further clues as to level. Moreover, it would not be a mere matter of chance that good percentages should be recorded by those authors who set little diagnostic and localising store by joint neurological examination, whereas those authors who pride themselves on being able to arrive at an almost entirely correct diagnosis on the basis of neurological data hold myelography in little esteem. One influences the other and the selection mentioned is also important in the sense that the "100% diagnostician" considers myelography to be superfluous in many clear cases, hence many well diagnosed myelograms will be absent from his statistics.

In judging the figures it is well to realise that mere "guessing" will produce an approximately 50% chance of diagnosing correctly or wrongly. If the percentages of correct diagnoses drop to below 50%, as in the following chart, it signifies that the examination was decidedly misleading. Admittedly, in 20% of correct diagnoses, for instance, the neurological diagnosis was completed in 20 individual cases with what may have been decisive individually valuable myelographic particulars; but against this – and it requires emphasis – the diagnostic insight was obscured, indeed erroneously guided, in 80%. The difference is not merely a quantitative one; errors in a high percentage of good diagnoses could be held to imply a certain inaptitude of the examination to correct neurological "guessing". The erroneous myelographic diagnoses in more than 50% constitute a decided dislocation of the insight already gained, since, instead of completing the neurological diagnosis, they upset it.

We estimated the *reliability of myelographic data after a preceding laminectomy* by means of the following classification, all the particulars being verified at

with signs of degeneration and in cases of subsequent persistence or recurrence of the symptoms.

We collected the following figures from the literature:

	Myelography correct
Barr (see Ståhl)	90%
Begg, Falconer, McGeorge (see Ståhl)	88%
Begg, Falconer, McGeorge (see Armstrong, p. 105)	78%
Borelli *et al.* (see Spiegel, 1957, p. 309)	85%
Epstein (p. 398)	85% – 90%
Key and Ford (see Lewin, p. 772)	72%
Kostić (p. 76)	60%
Krayenbuhl (p. 40)	86%
Krischek (p. 62)	61% – 70%
Lewin (p 772) – literature survey –	67% – 96%
MacCarty and Lane (see Spiegel, 1956, p 177)	89%
Munro (see Spiegel, 1957, p. 327)	50%
Roeder (p. 10)	66⅔%
Scoville *et al.* (see Epstein, p. 995)	65% – 75%
Ståhl	60%
Zukschwerdt (p. 115)	85%
Waris (pp 66 and 119)	68%

The following pronouncements on indication and reliability of myelography are more eloquent than figures:

Armstrong, p 105. " .in only 74 out of 95 patients (78 per cent.) with proved disc lesions did myelography show the lesion with accuracy, an error greater than one in five."

"Myelograms have.. certain disadvantages and dangers. The procedure is extremely unpleasant for the patient and is cumbersome and time-consuming".

". .should not be used... when the presence of a disc lesion is doubtful".

Barr (see Lewin, p 767): " in every surgical doubtful case..."

Biemond (1956, p 238) "Not certain, not necessary, not harmless", ".. therefore, only if specially indicated".

Epstein (p 395): "As one reviews the literature, not all are in accord with the opinion that myelography should be a routine procedure".

Epstein (p 398) "I am convinced of the importance of myelography before exploration of the spinal canal"

Harmon (see Lewin, p 767) In doubtful cases

Key and Ford (see Lewin, p 767) "When symptoms and findings indicate, a *negative* myelogram does not deter (their) recommending or carrying out operative treatment".

Kostić (p. 76) Not in neurologically clear cases

K [illegible] ulties.

[illegible]

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We estimated the *reliability of myelographic data after a preceding laminectomy* by means of the following classification, all the particulars being verified at

re-operation. (The material was studied again with the co-operation of the radiologist Van den Broek.)

		Myelography (II)					Myelography (I)
		Right	*Wrong*		*Right*	*Wrong*	*Right*
					+	—	+
True recurrent hernia at re-operation	51	28	23	=	55%	45%	74%
"Recurrent symptoms" (no hernia at re-operation)	76	15	61	=	20%	80%	70%
Recurrent symptoms: without signs of degeneration on the spine	24	4	20	=	17%	83%	80%
with signs of degeneration on the spine	52	11	41	=	25%	75%	67%
		43	84	=	34%	66%	71% (In 63 cases myelography (I) was known)
	127	127					

In a number of these groups we were able to trace the data relating to the primary myelography (I) which had contributed to the indication for the first laminectomy. Our material shows clearly how unreliably myelography reveals the condition in the spine upon which laminectomy has already been performed. The faulty diagnoses must be ascribed to the arachnitis, extradural adhesions, coalescence and scar formation, cystic formation in the field of operation, proliferation of the venous plexus, congenital malformations and (likewise reported) narrow dural sac, which were found at re-operation.

In six cases a recurrent hernia was found after the indeterminancy of the myelographic contours had seemed to rule this out on the supposition that it was merely a question of arachnitis, for instance. In other cases, however, the errors arose because contrast defects were ascribed to a true recurrence of a hernia which was found not to exist at operation.

The larger number of erroneous diagnoses in cases of backs without signs of degeneration, compared with those bearing such signs, is striking but understandable. It is reasonable to suppose that the discovery of these signs led the diagnostician sometimes to attach little significance to indeterminate defects in the contrast medium; when confronted with 'normal' backs, he was not thus misled.

The contrasting reliability of myelographies I and II is striking.

We find similar statements in the literature (Zukschwerdt, p. 168, Torma,

22.5 % wrong, p. 217, Knuttson, p. 60) in which the difficulty of interpreting myelograms after laminectomies is pointed out.

The following conclusions may be drawn from these surveys:

1. Primary myelography does not give certainty in any individual case.
2. Myelography after laminectomy produces useless information and is even misleading.
3. Myelographic diagnosis proves to be erroneous in 40–30–20–10 %, depending on several factors, including selection, indication and the condition of the spine.
4. It proved to be less reliable in cases of subsequent persistent or recurrent symptoms.
5. Primary myelography proved to be less reliable in cases of backs bearing signs of degeneration.
6. The general impression from records in the literature is that myelography should only be resorted to if therapeutic consequences depend on it; even then it should not be undertaken as a routine procedure, but only if specially indicated, as in doubtful cases.
7. It is evident from points 4 and 5 that myelography is least reliable when it is most needed.

Causes of erroneous myelographic diagnoses

Krischek (p 62)		*Zukschwerdt (p 115)*
17.3%	Recess, no hernia	10%
27.8%	No recess, but existing hernia	5%

Causes of erroneous diagnoses through myelography after laminectomy have just been mentioned.

In primary myelography these are:

1. *False positive* due to:

An unknown cause.

Venous plexus.

Posterior lipping (with or without more than physiological convexity of the disc).

Spondylarthrosis (owing to which the joints protrude into the spinal canal).

Osteoporotic arch.

Thickened roots (Epstein, p. 394; Lewin p. 771); (our own experience: *e g.*, pat. 271055).



Perineural root cysts.

Tumours (difference between hernia and cauda tumour is not always clear; de Grood, Ned. Tijdschr. v. Geneeskunde, 1950, p. 670).

Anatomical anomalies (Thurel, p. 26).

Developmental anomaly (partially sacralised 5th lumbar or partially lumbarised first sacral vertebra, Armstrong, pp. 102–103).

Congenital arachnoid bands (Lewin, p. 771).

Foreshortened lumbar sac (Bradford and Spurling, p. 70).

(Congenitally) narrow spinal canal, narrow dural sac (Epstein, p. 396).

Wide spinal canal.

As was explained on p. 179, the physiological concavity of the posterior aspect of the vertebral bodies – interrupted by physiological prominences of the disc – has a similar effect to puddles of water standing on an apparently flat stone (asphalt road). Owing to the breadth of the wide dural sac, the contrast medium is spread out and the prominence of the disc is therefore not covered. The lateral picture is the more unreliable; hour-glass recesses are sometimes seen on the antero-posterior (see illustration in Bradford and Spurling, p. 145, Fig. 65: false positive (negative exploration) associated with wide spinal canal (and broad dural sac)). (See p. 483, fig. C and *loc. cit.*, p. 179.)

2. *False negative* due to:



Lateral position of hernia; *e.g.*, in lateral recess under fan-wise extension of pedicle;

e.g., small hernia in intervertebral foramen (see p. 165) (Krayenbühl, p. 25, Briggs, Hirsch).

Exceptional aspect of hernia; *e.g.*, bud-hernia (see p. 166). A hernia can be as small as the head of a match, yet, owing to its position exactly under or in a root, may cause severe distress, though myelographically invisible on account of its small size.

E.g., necrotic nuclear tissue.

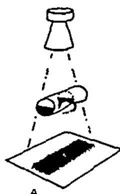
E.g., subclinical prolapses (Bradford and Spurling, p. 111).

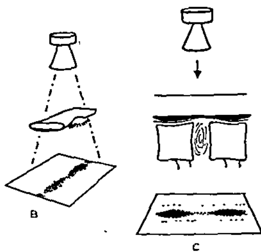
Anomalies, *e.g.*, hernia in a wide spinal canal beside a narrow dural sac, in which case the contrast medium does not approach the prominence and the shape of the column of contrast medium is therefore not changed. The spinal canal is often wider at L.V – S.I than at a higher lumbar level, which is why false negative myelography occurs more frequently at L.V – S.I (Ståhl).

E.g., a very narrow lumbar sac is nearly filled up by a certain quantity of contrast medium; a prominence reduces the diameter of the contrast column, but does not cause a defect (see Fig. A, p. 483).



Narrow dural sac in a wide spinal canal.
(Sacral perineural cysts)





E g., a very wide lumbar sac gives way in indeterminate contours to a prominence; the comparatively thin layer of contrast medium, together with the physiological prominence at other levels, displays such strange hour-glass defects that the "typical" configuration of a recess caused by a hernia is not recognisable (see Fig. B).

Another source of errors resides in the fact that, although correct evidence is produced, the supposed precision of myelographic examination is conducive to the drawing of incorrect conclusions.

A negative myelography for hernia may be falsely negative, inasmuch as an obscure hernia may nevertheless be present. Assuming that there really is no hernia, the diagnostician should not conclude from this that there is no other

abnormality, possibly treatable by surgery, or some other organic cause for the complaints and symptoms.

A positive myelography for hernia may be falsely positive. Assuming, however, that a hernia is proved at operation to be situated at the supposed level, who can tell whether the symptoms are due to this hernia or to one at another level, or to some other disorder? This can only be established with fair certainty by monoradicular mechanical stimulation of the root involved under a local anaesthetic.

Example:

Myelography L.V – S.I positive (L.IV – L.V negative)
Operation: Hernia L.V – S.I.
Stimulation S.1: No typical pre-operative pain.
Stimulation L.5: Typical pre-operative pain.
Exploration under L.5: *Small bud-hernia.*

This can be explained on the basis of statements made in the literature to the effect that a hernia need not necessarily produce symptoms (Armstrong, p. 66, Lewin, p. 767, Roeder, p. 3, Krischek, p. 64). This tallies with Andrae's findings (see Schmorl, p. 155, Zaaier, p. 177). At autopsy he found prolapses of the discs in 15 % of the spines he had examined and which had been symptomless. Trowbridge and Frenche (p. 339) applied myelography to 25 healthy persons (no low back pain, no radicular symptoms) and found 14 recesses caused by herniations.

Summing up the foregoing, it may be said that the reliability of myelographic examination has proved disappointing, while in an individual case myelography does not produce conclusive evidence. Although Epstein says (p. 398) "Myelography... compares most favourably with other diagnostic procedures", there are probably few methods of examination which detract from neurological insight in so speciously exact a manner as does myelography of the lumbosacral canal.

Having thus tried to estimate what benefit the patient derives from an examination of the kind, we should now enquire into the disadvantages it entails to him or her.

Not infrequently, the patient will say after the operation that the surgical procedure was simple compared to the making of the myelogram (Spurling, p. 92).

The subjective inconvenience and distress, the number of days the patient has to be under observation – exceeding those needed for the remainder of the examination and fluid analysis – hence the *extra loss of time*, depend on the method employed.

The *dangers of radiation* have already been discussed. Every myelography necessitates fluoroscopy for two to five minutes, depending on the method, and longer still according to some investigators. Without screening, either for direct evaluation or to determine the site and moment for the exposures that are to be taken, myelography is useless. The intensive radiation to which the gonads are exposed during lumbo-sacral screening has already been mentioned. Assuming that X-rays have an injurious effect, the quantitative extent of which is not yet known, any risk not strictly necessary should be avoided. Seeing that, with five minutes' fluoroscopy for myelography, the testes are irradiated with an intensity equal to the scattered rays of 5 times > 12,000 = > 60,000 skull photographs (!), the first of the routine procedures to be scratched off the list should surely be the myelography, which is unnecessary.

Complications resulting from the contrast medium introduced have been described by many authors. They vary from harmless, passing disorders to less harmless ones, *viz.*, arachn(oid)itis leading to invalidism, with lethal complications at the end of the scale (*e.g.*, through aqueous contrast media). Ziedses des Plantes (p. 1196) points out this danger. It was not until lipiodol had been used extensively as a routine procedure for roughly ten years that



Ethiodan in root sheaths
two years after myelography

its injurious effects were discovered. Even then it was not understood why the gravest forms of arachnitis occurred, particularly after opening a dural sac containing lipiodol. In view of the consequences of using old lipiodol which had been exposed to the air, this complication was ascribed to the effect of air upon the lipiodol in the opened dural sac.

Although no disturbing rumours are circulating, we still do not know whether or to what extent the so-called re-absorbable oils and aqueous contrast media will prove to be deleterious in the future. There are reports in the literature to the effect that these oils remain unabsorbed and visibly mobile to some extent in the dural sac for years (Epstein, 409: pantopaque, 9 years). In point of fact, the re-absorbability of these oils is said to be no more than ostensible. Krump (p. 460) supposes that the contrasting iodine is split off and re-absorbed, but that the oil, no longer demonstrable radiologically, remains behind. Those who ascribe persisting low back pain, or a heavy feeling or pain in the coccyx, to residual contrast oil in the tip of the lumbar sac are probably not far wrong.

Epstein (p. 408), Ernsting (pp. 92 and 99), Krump and Albrecht (p. 449) and Zukschwerdt (p. 114) report on the drawbacks and complications.

Pantopaque pulmonary embolism, 4 cases:	Ginsburg *et al.* (see Spiegel 1956, p. 177). Epstein (p. 410).
Contrast oil in the ventricles and at the base of the skull	Epstein (p. 408), Krump (p. 460), Zukschwerdt (p 114). Our own material: 3 cases.
Neuritis optica through oil at the base of the skull	Our own material: one case.

Can these objections be overcome by *getting rid of the oil contrast medium?*

1. Its elimination has to be directed by screening, which increases the dangers of radiation further.
2. When oil-arachnitis is discovered at operation, droplets and filmy adhesions are seen, but most of the oil by far is held in encapsulated cysts between the roots. As Prick and Hoefnagels have shown, a large amount of oil favours inflammation, but only a small amount of oil is needed to induce the adhesions themselves.
3. Complete evacuation appears to be impossible; enough always remains to set up arachnitis.
4. Elimination during operation by opening the dura is a rigorous measure and, even so, complete evacuation cannot be effected.

5. The roots might be damaged while aspirating with a needle under X-ray control.

After going over the complications possibly attendant upon lumbar myelography in great detail, Krump and Albrecht (1956) conclude as follows: "Nur durch die Anwendung einer äuszerst strengen Indikation zur Myelographie, kann erreicht werden, dasz die Anzahl der Patienten die unter eventuellen Spatfolgen zu leiden haben, auf ein Mindestmasz beschrankt wird".

It will be obvious why we subscribe, personally, to the opinions gleaned from the world's literature regarding the indication for lumbar myelography. Indeed, in view of the comparatively large measure of its unreliability, coupled with the comparatively serious objections cleaving to it, we ask ourselves with amazement how it comes to be that myelography as a *routine* procedure in the examination of back disorders has not been generally condemned as a professional error. The position has not been improved by the use of re-absorbable oils and aqueous contrast media.

By condemning myelography (as a routine method) we are disarming ourselves, particularly as we have already referred to the comparative unreliability, both for diagnosis and for locating, of the data obtained from neurological examination. If we estimate myelography to be reliable in 70% of cases approximately, the same percentage would apply, approximately, to the clinical symptomatology of diseases of the back. We even suspect that it is this very dilemma which drives many a diagnostician in despair to myelography, with the result that the clinician, as verification of both sets of findings showed, continued to flounder in the same quandary.

Approaching the indication for myelography from a practical point of view, the importance of taking the patient's whole personality into account again becomes plain; plainer still when it comes to an indication for surgical intervention. This will be dealt with more fully in a subsequent chapter.

Going by his experience of negative explorations, recurrency operations and faulty myelograms, the author, as a neurosurgeon, believes the following may serve as a *practical guide in deciding upon indications for myelography:*

(1) Tentative psychiatric-neurological-internal examination.

(2) Lumbar puncture and cerebrospinal fluid analysis.

If significant changes of any kind (hence including psychiatric changes) are found, apart from those belonging to the neurological L.5, S.1 or S.2 syndrome, the patient should be classed under the heading "Special indication".

(3) Definitive neurological examination.

If, in the psychiatrist's opinion, the complaints are founded on fact and the clinical-neurological symptoms point to a radicular syndrome of root L.5, S.1

or S.2, the patient qualifies in principle for surgical exploration of the L.IV – L.V and L.V – S.I levels, the terms of the diagnosis being "Hernia or similar lesion". In cases of doubt as to the existence of a change at L.III – L.IV (L.4 syndrome), the patient is placed in the "Special indication" group.

(4) Antero-posterior and lateral tentative radiological examination.

From this, facts may come to light, such as spondylolisthesis, which call for further deliberation. If the spine is obviously a transitional one, it is necessary to reconsider whether the neurological symptoms, interpreted in the light of transitional tendencies, are sufficiently clear as to location for a lesion at one of the two lowest lumbar levels. If not, the patient is again relegated to the "Special indication" group.

Intervertebral narrowing, posterior lipping or a local spondylosis at the anterior aspect of the vertebral margins may be helpful in level diagnosis. If the radiological and neurological diagnoses are flagrantly contradictory and the mutual contradiction cannot be resolved by simultaneous exploration of the levels L.IV – L.V and L.V – S.I, the patient must be placed in the "Special indication" group.

(5) In the absence of a special indication, we advise

Operation in an acute stage.

Local anaesthetic.

Exploration L.IV – L.V *and* L.V – S.I (*and* under S.2).

Monoradicular stimulation of the roots for confirmation and to decide whether or not extension of the surgical treatment is indicated.

"Special indications" for myelography

(a) Severe, persistent, subjective distress resisting conservative treatment without neurological symptoms.

To be differentiated from psychogenic disturbance.

(b) Protein content of the fluid 0.050 $^0/_{00}$.

To be differentiated from (cauda) tumour.

Hernia at high lumbar level.

Some other neurological abnormality.

(c) Internal changes suggesting metastases; *e.g.*, distinctly, though inexplicably, raised blood sedimentation rate.

(d) L.III – L.IV, L.II – L.III, L.I – L.II syndromes, L.4, L.3, L.2 syndromes respectively.

(e) Transitional vertebra with unclear neurological syndrome of insufficient localising value.

(f) Glaring contradiction between neurological and radiological evidence.

E.g., disc narrowing between L.V and S.I with local arthrosis associated with syndrome of L.4.

E.g., vertebral lesion (traumatic) at high lumbar level associated with syndrome of L.5 or S.1.

"*Contra-indications*" *for myelography* (excepting special indications)

(a) Clear radicular L.5 or S.1 syndrome with radiologically normal spine. Myelography is unnecessary.

(b) Previous laminectomy. Myelography is misleading.

(c) Extremely wide spinal canal. Myelography is exceedingly unreliable.

We have tested the utility of this plan in practice. In one hundred laminectomies we did *not* make full use of the data available before the operation; that is to say, we only made use of the data in accordance with the above plan, and in that chronological order. This was permissible, because we *knew* in these cases that all particulars, including myelography, had been verified by experienced specialists working with us regularly. They would certainly have warned us if anything out of the way had occurred. Thus the laminectomies were performed following the diagnosis of a radicular syndrome of L.5, S.1 or S.2, pathological changes at higher lumbar levels being ruled out on the basis of the clinical history, neurological and tentative radiological evidence. In this way we approached ideal conditions for a test, since the results of the complete routine examination and those of the above plan could be compared, as far as diagnosis, indications and level diagnosis are concerned, in one and the same patient.

In these patients, diagnosed as suffering from some abnormality at one of the two lowest lumbar levels, the L.IV – L.V and L.V – S.I levels were explored simultaneously by monoradicular stimulation of the roots concerned. At post-operative verification, the records of these patients differed in no way whatever from those relating to operations performed upon the evidence of what was at one time the usual complete examination. It should be added, however, that in 21 % a wrong level would have been explored if, depending on myelography, we had decided before the operation to expose only the one level indicated by the myelogram.

We have no experience of *canalography*, the method and advantages of which are described by Knutsson, Kramer, Van der Werff and Prick * Moreover, it is our impression from the literature that, up to the present, few have been in a position to judge [illegible] ation.

Dis[illegible] puncture of a disc [illegible] show up the

* Luyendijk and Del Prado 1959

site of a rupture in the annulus fibrosus. The method was introduced, severely criticised and passionately defended, but did not find general favour. We do not think we are being premature when we say that, judging by reports in the literature, it has already been abandoned. Having no experience of it ourselves, there is no point, under the circumstances, in referring to other authors' opinions as to its merits and demerits. Chief among these are the painfulness of the treatment and the risk of injuring nerve tissue or initiating herniation through the puncture of a pathological disc under tension. Arguing along theoretical lines, we should only wish to ask how the diagnostician knows *which* disc is diseased and has, therefore, to be punctured? Assuming that in 30% of typical hernia cases the clinical diagnosis is unable to determine the right level and allowing for multiple disc lesions in 15%, this means that, with a single puncture, a healthy disc is lanced in 25 out of 100 cases (30% of 85) and is maltreated with contrast medium, the influence of which upon the biological processes in the disc is unknown to us. If, however, in order to make sure, two lumbar discs are punctured, it can be roughly calculated that in every hundred cases approximately 85 healthy discs are thus victimised. Even without taking other objections into account – the principal one of which being that the sole purpose of the procedure in question, for so intricate a complex of complaints and symptoms as the diseased back, seems to be to discover a hernia nuclei pulposi – it does not surprise us, therefore, that the method has virtually been abandoned.

ADDENDUM TO CHAPTER 6

The mechanisms which induce the syndromes. Predilection for the low lumbar level. Classification of the syndromes.

Many questions bound up with the symptomatology of back disorders, not yet discussed, now call for some attention.

Mechanisms of radicular syndromes

A few of the following arguments have been touched on in the preceding pages; they will now be recapitulated together with some other points of view.

For a long time the accepted explanation was the incursion of an inflammatory factor, either or not in conjunction with compression of the root. A *mechanical* impediment cannot be the only cause of disturbances, since some root compressions occur without producing symptoms, whereas in other cases the same impediment does produce symptoms, either transitory or persistent.

Before hernia became a commonplace in the neurological world, "*sciatic neuritis*" was the generally accepted diagnosis for clinical pictures which would now be described as a radicular syndrome resulting from, say, a hernia (Kinnier Wilson, p. 350). "Transitional neuritis" (neuritis due to irritation of nerve tissue caused by a transitional vertebra) is another term dating from that time. This does not mean to say that an infectious lesion of a big nerve cannot occur

as a clinical picture in its own right, similar to that described recently by Fabregoule and his colleagues as a complication of diabetes.

The general condition of the patient (blood, spinal fluid, temperature) does not betray signs of inflammation. In 45 % of hernia cases verified at operation, the sciatic nerve was painful on pressure along its course (Krayenbühl, p. 36), hence by no means proves the existence of sciatic neuritis.

Monoradiculitis is far more likely to be responsible for radicular syndromes, in which case the inflammation would be an adventitious factor in an already existing disorder (compression, hyperaemia, cold, allergy) (Cossa, p. 218, Krischek, Irsigler, Reischauer, pp. 8–11, Roeder, p. 5, Veraguth, p. 173). This view is not very satisfactory, as the supposed inflammatory changes were only demonstrable in a few cases, and then not convincingly (Krischek, Bozsik, our own investigations: Wyers, see pp. 493, 300). Primary adhesions of roots found at laminectomy need not be due exclusively to inflammation of the root; local inflammation or haemorrhage in the spinal canal, or changes in the tissue due to pressure may produce the same result.

Dandy's opinion (1941), to the effect that sciatica is produced only by a tumour of the cauda equina, disease of the spine or hernia, is not shared by all authors.

Some of the opinions expressed are recorded briefly below.

Armstrong (p. 159). Vast majority of patients once regarded as suffering from "primary" sciatica had in fact a lesion of one of the lower lumbar intervertebral discs.

Bradford and Spurling (p. 102) "Primary sciatic neuritis is truly a clinical curiosity". (p. 128). A sciatic syndrome caused by a hernia without a neuritic component is probably likewise rare.

Krayenbuhl (p. 31): "Nicht Druck allein lost die Schmerzen aus – sondern... eine symptomatische Entzundung der Wurzel".

Granit *et al.* (p. 125): Internal, so-called structural changes in the nerve tissue produce "artificial synapses" so called, thus impulses in a motor fibre may cause short-circuiting on sensory fibres and, therefore, pain.

Reischauer (p. 11). "Reine Neuritis ohne Mechanisches ist Ausnahme".

Roeder (p. 19): "Eine isolierte primäre Neuritis (ischiadica) ist selten".

Krischek (pp 9–12): As stated elsewhere, signs of neuroradiculitis are not clearly demonstrated, though that is the aim of his monograph.

An investigation pursued by Lorentz de Haas (see Biemond, p. 241) is interesting and instructive. He made a careful enquiry into the fate of those patients who, before the hernia period, had been diagnosed as suffering from "sciatic neuritis" and found that this diagnosis had been revised in the vast majority of cases and that many had since been found to have a hernia. Zukschwerdt (p. 93) enters into this matter fully, mentioning the opinions of many authors:

"Die hauptsächliche Ursache des monoradiculären neuralgischen Schmerzes ist mechanischer Natur". The abrupt cessation of radicular symptoms, perhaps after chiropractical treatment, is, he thinks, an argument against the existence of factors of a more chronic nature (mechanical neuritis, hyperaemia, p. 94, p. 153). He ascribes the hypersensitivity to cold to a pre-existing lesion of the root or nerve (p. 98).

When studying the literature, especially German literature, it should be borne in mind that the word "neuralgia" (Landouzy, p. 875) is often used in its literal sense, *viz.*, pain in a nerve (Krischek, p. 7, Roeder, Zukschwerdt). In the discussion of radicular pain, as is meant by sciatica, one sometimes finds a comparison with neuralgia major nervi trigemini, for instance; the radicular pain associated with cases in which no lesion of the root is found at operation is thought possibly to be of the same nature as intercostal neuralgia, for example.

Pedersen and his colleagues (p. 388) wonder whether radiating pain can be interpreted as referred pain in the anterior rami (to the extremities) as the result of irritation of the posterior rami and the sinuvertebral nerves (at the site of the joints and in the spinal canal), provided that the painful stimulus is strong enough. Vernon (see *ibid.* p. 391) then speaks of "(referred) reflex radiation".

It is, however, clear that other factors as well are capable of inducing radicular symptoms.

Armstrong (pp. 53 and 65) speaks of *deformation* and adhesions.

Lindblom and Rexed (p. 431) proved macroscopically and microscopically that "compression (hernia and osteophyte) was shown to have caused serious *injury* to the nervous structures" (degeneration, regenerative processes, deformation, atrophy).

Lewin (p. 633) and de Sèze (p. 1788) think it is not impossible that a nerve root may suffer impairment of its *vascular supply*.

Some authors connect *oedema* causally in varying degrees with a radicular syndrome (including Armstrong, p. 64, B. Brouwer, pp. 440–441, Irsigler, p. 482, Goslings – *e.g.*, oedema of ligaments, p. 2014, Thurel, pp. 19, 28 and (1952) p. 9, Veraguth, p. 173, Voris *e.g.*, after lipiodol myelography, p. 116). Others, on the other hand (Zukschwerdt, p. 88), consider the influence of oedema of the root and adjacent structures to be "conceivable, but unproved". It is said to occur in the course of allergic processes, in several roots, but to manifest itself clinically in a root already compressed.

These quotations could be multiplied by many more. Roughly speaking, the recognition of a mechanical factor as the cause of a radicular syndrome predominates. A mechanical irritant, reminiscent of inflammation, is thought to be a possible component, though lacking proof. Oedema is found, but can scarcely

be accepted as the cause of an abrupt functional disturbance; at most it may be an accompanying symptom, yet radicular syndromes are known to occur suddenly.

It is interesting to note that microscopy is inconclusive in this matter.

Armstrong (pp. 158, 53): Irritation, oedema, fibrosis; no "itis".
Borsik (p. 255, also see *loc. cit.* p. 300): Degeneration, but no neuroradiculitis.
Irsigler (p. 485): Symptomatic inflammation through mechanical injury, acute and chronic infiltrations, degeneration; or no histological changes. (N.B. The particulars derive from the examination of a piece of *intradural* root.)
Krischek (p. 12); see *loc. cit.* p. 299): Inflammatory reactions in root after rhizotomy for hernia.
Lindblom and Rexed (p. 426):

No inflammation Deterioration of nerve tissue Arachnoidal proliferations No histological findings N.B. Unselected autopsy material	23 cases of nerve root compression associated with hernia

Norlén after survey of literature, p. 19: "True to say we lack a wholly convincing pathology of interstitial neuritis".

The result of the microscopic examination (Wijers) of a number of sensory roots obtained by extradural rhizotomy (between dural sac and ganglion) was as follows:

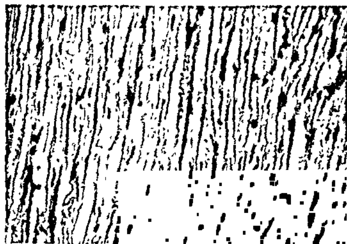
Rhizotomy with hernia	Rhizotomy with negative exploration for hernia	
	without root thickening	with macroscopic root thickening
5	17	3

In none of these three groups were there clear signs of active inflammation. Sometimes there was slight oedema and in one case there was a trifling increase of connective tissue in the roots.

Such changes in nerve tissue as were observed proved to be artifacts. In the proximity of the incision, the myelin ran out of the sheath, or was compressed by the operation trauma, owing to which swellings were seen, especially on either side of Ranvier's nodes.

The presence of inflammation infiltrate in the root was not detected in any of these cases. But an accumulation of the white corpuscles was seen on the inner wall of the blood vessels, and this could account for the inflammatory irritation set up by the operation (dilatation of the vessels and decelerated circulation of the blood). Any irritant which slows down the circulation of the blood can do so within a few seconds. The phenomenon occurs equally in

Text continued on p. 497.



Artifacts produced by surgical lesion. Cork-screw shaped, retracted axis cylinders, granular break-down of myelin.



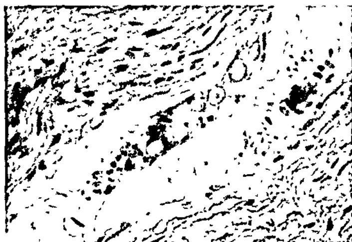
Artifacts in extradural sensory root, pseudo-oedema, vacuoles Surgical lesion close to the place where section has been made myelin has partly disappeared, with fragments still adhering to the axis cylinders, a few *Ranvier constrictions* are *still visible. No reactive* symptoms of inflammation



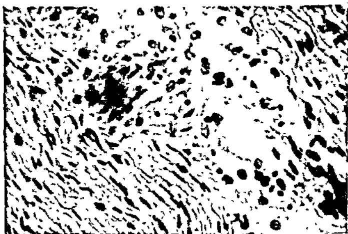
Transverse and longitudinal section of myelinated fibres. Towards the middle, the transversely cut fibre bundle shows thickened fibres, local thickening of the medullary sheath and axis cylinders close to the plane of section. Artifacts due to surgical lesion.



Blood vessel with peripherally situated leucocytes. No infiltration in the surrounding tissue.



Oedema?
No cells outside the blood vessel.



Pseudo-oedema in thin wide-meshed perivascular connective tissue due to tearing.



Peripherally arranged leucocytes, perivascular pseudo-oedema. (No exudation of white corpuscles.) Wavy axis cylinders.

normal healthy tissue exposed to irritation of a like kind; hence these groupings of cells in the walls are to be differentiated from inflammatory infiltrates already present prior to the extirpation.

Sometimes perivascular (pseudo-)oedema was encountered, but in many cases it was possible to show that this was due to the tearing of the very tenuous perivascular tissue. This does not bear out Irsigler's view (p. 482) that the thickness of some roots is always due to oedema, although the pathologist would, of course, have to admit that the absence of demonstrable oedema in sections does not provide certain evidence of its absence *in vivo*.

Occasionally trivial perivascular accumulation of fluid was seen, probably derived from vascular tearing, but the fibres were never seen to be pushed apart by oedematous swelling.

The pathologist, having no foreknowledge of the macroscopic aspect of the roots, found no difference between the microscopic aspects of the thick and thin roots.

As we found no signs of inflammation, infiltrate or undoubted oedema, we think the radicular symptoms can only be explained by abnormal anatomical relations (compression, etc.). We do not know whether swelling – which, to or mind, is *not* demonstrably oedematous – accentuates the effect of an initial compression.

In all radicular syndromes there is some connection or interplay between the primary space-occupying process, the constitutional or "conditional" spaces available and the complicating factors, if any (such as reactive inflammation, venous congestion), on the one hand, and hyper-irritability of the nervous system and the patient's psyche, on the other. The multiplicity of factors involved accounts for the relativity in respect of the moment at which a radicular syndrome presents its varying clinical picture and its severity.

Mechanism of low back pain

The same facts as those just mentioned are involved, *mutatis mutandis*, leading to a reaction between constitutional/conditional factors and adventitious conditions. To this we may add the following.

We know of no "cause" of back complaints – nor, indeed, of a radicular syndrome – which could also be present without producing these complaints or symptoms, while, on the other hand, those same complaints and symptoms may be present quite obviously in the absence of the suspected cause! (As far as spondylarthrosis is concerned, this may be assumed to be known; hernia nuclei pulposi is another good example: Armstrong, p. 65, Bradford and Spurling, p. 104, Andrae, see Schmorl, p. 155, McRay, see Spiegel 1957, pp. 308 and 326· "Nearly everybody 40 years of age or older has at least one posterior cervical and one posterior lumbar disk protrusion" – post mortem investigation by dissection.) This fact implies that the word "cause" is really used erroneously, also that *the* cause of low back pain is not known (or does not exist); low back pain is the resultant of an interplay between predisposing and precipitating factors. These may be subdivided as follows:

1. Low back pain *without* a known anatomical substrate. The (lumbar) spine is the apparatus for movement and support of the body. Any incongruity there may be between the demands made upon this function and the work the spine itself is capable of doing, has to be compensated for as best it may and as long as possible by the systems of long and short back muscles. When these muscles become exhausted, this in itself produces pain and, before all, pathological lassitude

2. If the muscles fail chronically in their task, this incongruity between demands made and performance widens, hence the dysfunction of the spinal column becomes chronic. As a result, the discs, joints and ligaments are exposed to a pathogenic strain and pain is produced through the action and reaction of opposing forces Clearly, the poorer the quality of the muscular system laid down (asthenia, dystrophia musculorum progressiva, leptosome habit, etc.)

and the more this inferior disposition is represented in weakness of the ligaments (asthenia) and other parts of the skeleton, the sooner, relatively, will that system fail. This accounts for the characteristic predisposition of the endogenously weak back to low back pain. This pain will be set up all the readier if excessive demands are made chronically upon the skeleton and the muscular system. Such demands may be made – though we think in exceptional cases – irrespective of the patient's mode of life, but mostly they will be due to endogenously determined awkward motor behaviour and often the patient injudiciously embarks upon strenuous activities wholly unsuited to his disposition, or for which, through lack of training, he is not yet, or else no longer, fit.

Lastly, for lack of insight, want of energy, through the effect of an early descending line of life upon bodily manifestations as well as for other reasons, precisely those patients who most need to, make no effort to keep themselves physically fit. We are convinced that sub-standard intellectual abilities and temperamental shortcomings are likewise determined endogenously in this respect.

3. Low back pain *with* a known anatomical substrate. Local organic changes in joints and ligaments are said to irritate sensory nerve endings (Hirsch, see Lewin, p. 867, Lewin, p. 633, Steindler). Lindblom induced low back pain by injecting saline into the discs. Bradford and Spurling, p. 112, refer to a synovitis of the joint. The consensus of opinion is that low back pain, resulting from whatever lesion (trauma, tumour, infection), whether primary or derived secondarily from a morbid change elsewhere in the body, is induced by irritation of the sinuvertebral nerves (ramus meningicus nervi spinalis). (Armstrong, p. 57, Ernsting, p. 33, Bradford and Spurling, pp. 20 and 104, Hagelstam, p. 26, Steindler, Pedersen *et al.* p. 377.)

Although it is by no means an established fact that nerve tissue runs in the annulus fibrosus, no departure is made from this rule for the manner in which a disc lesion, with or without herniation, is said to produce low back pain. Hirsch (see Spiegel 1957, p. 332) denies that fibres run in the annulus, nor will he admit that "backache is the result of disc degeneration". Even if the annulus should be innocent of nerve fibres, it is reasonable to suppose that protrusion of the disc will so disorganise the surroundings of the intervertebral disc, particularly the posterior longitudinal ligament, that irritation of the nerve fibres, which do invest this area undoubtedly, must result.

Pedersen makes it clear that the posterior branches of the peripheral nerve supply the spine, notably joints, ligaments, etc., on the outside of the intervertebral foramen. Irritation of these little nerves would set up some of the low back pain.

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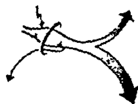
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1. The radicular syndrome in the distal innervation areas of the peripheral nerve, and
2. "Radicular low back pain", the radicular syndrome of the ramus medialis dorsalis.

For the above reasons it is suggested that possibly the areas to which radicular pain radiates may be arranged in the aforementioned pattern of the peripheral nerve. This would mean that the ramus medialis dorsalis, which supplies the immediate surroundings of the intervertebral foramen, might likewise serve as a radicular radiation area; and the same equally applies to another side-branch of the peripheral nerve sent off earlier still, *viz.*, the ramus meningicus nervi spinalis. The fact mentioned above, that radicular symptoms often, especially at first, are confined to proximal areas closely adjacent to the spinal column, argues in favour of the view that "radicular low back pain" occurs before all other radicular symptoms in the bundles of nerves (ramus medialis posterior and ramus meningicus) lying closer still to the spine. Irrespective of the aetiology, any compression of the nerve tissue in the intervertebral foramen might evoke such symptoms. The fact that pressure-raising factors (coughing, sneezing, etc.) likewise induce or exacerbate low back pain is certainly a further point in favour of the possibly radicular genesis of some kinds of backache.



It remains an open question why radicular pain first radiates to the proximal branches of the peripheral nerve. But we would refer to the comments on the influence of background activity upon the occurrence of neural loss symptoms preferably in the distal parts of the peripheral innervation areas (p. 373). We suggest a comparable, though reverse, effect, *viz.*, high background activity from parts of the peripheral nerve situated close to the ganglion carries across the pain threshold those mild pain stimuli which result from an initially minor lesion of the corresponding nerve fibres in the root.

Seen in this light, we subscribe to the view that some part of low back pain may be of a radicular nature, meaning more specifically the diffuse low back pain at a lower level than the site of a root lesion found subsequently, especially in the region of the coccyx and of the sacrum.

The varying aspect of the clinical syndromes

The diversity of the factors mentioned above is responsible for this variety. It is a known fact that diseases of the back may begin with low back pain

4. Without contradicting the foregoing, we suggest that there is a fourth possible way in which low back pain may arise, basing this supposition on the following facts:

a. Hagelstam (p. 26) shows that one and the same disc lesion induces both the low back pain and the radicular pain associated with the syndrome. As a matter of fact, this has become a commonplace and to it are ascribed the merging of the one syndrome into the other, the varying picture and other phenomena.

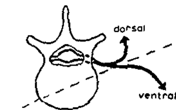
b. We have been able to verify in our patient material that the local tenderness on pressure is revealed by the patient, in the vast majority of cases, as being at one level or several levels lower. This concords with the anatomy of the sinuvertebral nerves as reported by Roofe.

c. We have noted that manipulations around the ganglion in the intervertebral foramen soothed the pain at the same level and a little lower in the spinal canal; *i.e.*, whereas the corresponding area of operation was painful on being irritated, this was no longer so after such treatment, during which, nevertheless, the ramus meningicus nervi-spinalis must obviously have been damaged.

d. Low back pain is a rather general term for pain intimated by the patient pre-operatively and post-operatively to be felt in a vaguely-defined area. On being closely questioned, however, the patient is sometimes brought to state that the level and character of the pain vary. It is therefore not a rash inference that low back pain has several components.

e. Our records show that in 23 % radicular pain radiated to distal innervation areas, whereas in 65 % it was located in the lumbar region, the hip and the buttocks. Hence there are several components in a radicular syndrome.

f. Let it be remembered in this context that Kuhn demonstrated the topographical arrangement of the filamenta radicularia in laboratory animals and that we established a similar arrangement in the root, right in front of the ganglion, in man.



g. Finally, a strong argument in favour of this view inheres in the known anatomical data which Bolk was the first to describe and probably more clearly than anybody. On the basis of his dissections he declared (p. 16) that there was a primary division of the peripheral nerve into a dorsal and ventral branch. He predicted on theoretical grounds that some time separate syndromes would be described, marked off by the division of the body into a dorsal and a ventral sphere of influence for these respective nerve branches.

On this hint we distinguish between:

in pushing a protrusion back with force, and this is the experience of several other authors (Spurling, 1958, p. 49).

Summing up the foregoing in a few words, we consider that the varying neurological picture, the intermittent severity and the periodically changing radiation to different areas are caused by more than one aetiological factor.

The varying picture will be brought about by the combined action of, possibly, a protruding disc, the available space in the spinal canal determined constitutionally and by the condition, the degree of oedema or other complications in the roots and adjacent structures, while some part may be played by other constitutionally and "conditionally" determined circumstances, such as allergies and metabolic processes. Furthermore, incidentals may intervene, like changes in the macro- and micro-climate, the rest factor and emotional situations.

Multiple syndromes – Multiple hernias

Simultaneous hernias at L.IV – L.V and at L.V – S.I have been found in the following percentages:

Ursula Clinic	12%
Armstrong (p. 205) records	10% to 20%
Kostić records	3% to 4%
Love and Walsh (p 101)	12%
Spurling (1958, p 32 in a review of the literature)	0 to 18%

Multiple syndromes, in the sense that the clinical picture points to the lesion of more than one nerve-root, may be brought about by:

1. Multiple hernias.
2. One hernia compressing two roots at the same level.
3. The multi-radicular character cannot be other than apparent if, through variations (anomalous transitional fibres), the lesion of one root is reflected in more than one of the root areas established by clinical experiment.

The changing picture of a clinical syndrome may derive from exacerbations of multiple herniations in successive stages. The mechanism of functional listhesis teaches us to expect a second disc lesion, with or without herniation, after laminectomy or spondylodesis, if the muscular system is not properly trained to cope with the resulting instability, or is fundamentally incapable of doing so.

which disappears, however, suddenly upon the onset of the radicular syndrome. The obvious assumption is that a corresponding structural change took place in the lesion; then, if a hernia is found during subsequent surgery to rectify a radicular syndrome, an equally obvious assumption would be that this change consisted in the sudden rupture of the annulus fibrosus.

Variations in the neurological aspect, such as its episodic nature, the cessation and recurrence of neural loss symptoms, might be ascribed to displacement of the bulging nuclear material, now moving into an unfavourable position in relation to the root, then shifting again and leaving the root free. However, this quite plausible explanation does not by any means always hold good. Prolapses are found at operation at a time when the neurological symptoms have again remitted, although the prolapse is still distinctly compressing the root. It then occurs to one that possibly adventitious factors had previously and transiently changed the nervous system. Another possibility to be considered is that the function of a root has been so radically cut out, that pain is no longer induced by stimulation.

It is a known fact that the effect of a strong stimulus submerges the perception of a pre-existing, weaker stimulus. If the hypothesis of the radicular genesis of low back pain by an initial mild pressure on the nerve-root be accepted, it might be conceded that, at a worsening stage of the malady, a sudden, far more severe pressure on a larger mass of the root could well set up such violent pain radiating to distal parts of the area that it would completely overshadow the earlier low back pain. This might be yet another reason for the changing picture of the clinical syndrome.

Armstrong (p. 62) offers a complicated explanation for this. Bradford and Spurling (p. 105) point to the varying degree of oedema and also assume that there are remissions in the protrusion of the nuclear material. Brocher (p. 25) also holds several factors responsible for the varying nature of a disc lesion and stresses the fact that herniation is only one manifestation of the whole clinical picture.

We should not forget that the patient is ill *in toto*, even if the symptoms of the back and lower extremities dominate the picture. In this connection Zukschwerdt points out that in many clinical histories low back pain goes along with brachialgia and cervical migraine.

We find it very difficult to believe that the changing course of events could depend entirely upon the degree of extrusion of an intervertebral disc. It may possibly account for acute exacerbation, but, like Armstrong (p. 64), we think it is highly improbable that completely extruded nuclear material could slip back again spontaneously and acutely. We have never succeeded at operation

in pushing a protrusion back with force, and this is the experience of several other authors (Spurling, 1958, p. 49).

Summing up the foregoing in a few words, we consider that the varying neurological picture, the intermittent severity and the periodically changing radiation to different areas are caused by more than one aetiological factor.

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Simultaneous hernias at L.IV – L.V and at L.V – S.I have been found in the following percentages:

Ursula Clinic	12%
Armstrong (p. 205) records	10% to 20%
Kostić records	3% to 4%
Love and Walsh (p 101)	12%
Spurling (1958, p 32 in a review of the literature)	0 to 18%

Multiple syndromes, in the sense that the clinical picture points to the lesion of more than one nerve-root, may be brought about by:

1. Multiple hernias.
2. One hernia compressing two roots at the same level.
3. The multi-radicular character cannot be other than apparent if, through variations (anomalous transitional fibres), the lesion of one root is reflected in more than one of the root areas established by clinical experiment.

The changing picture of a clinical syndrome may derive from exacerbations of multiple herniations in successive stages. The mechanism of functional listhesis teaches us to expect a second disc lesion, with or without herniation, after laminectomy or spondylodesis, if the muscular system is not properly trained to cope with the resulting instability, or is fundamentally incapable of doing so.

Predilection for the lower lumbar region

If a hernia develops, why does it do so at L.IV – L.V or L.V – S.I in the vast majority of cases?

Among the material of the St. Ursula Clinic we found hernias at:

L.IV – L.V	L.V – S.I	L.III – L.IV	Multiple
46%	39%	3%	12%

Comparable figures are reported unanimously in the literature. Why this partiality of hernia for the two lowest lumbar discs? We should not forget, however, that low back pain also occurs at the site of these two vertebrae *without a demonstrable hernia. To those who are inclined to reduce all* pathology of the spine to that of the intervertebral discs, the obvious conclusion will be that in those cases, too, the low back pain will be due to an overlooked disc lesion, whether complicated by a herniation or not. To us such a conclusion would seem premature, for, theoretically, the course of events might well be just the reverse, *viz.*, the lower part of the lumbar spine might be "diseased" and, under certain circumstances, that disease might cause secondary degeneration of the disc.

We favour the latter supposition for reasons to be explained and intentionally transpose the question put as to the predilection of a hernia for the two lowest lumbar discs to another question: Why, then, is it precisely the lower part of *the lumbar spine that is so often diseased?*

1. First of all, this is only an apparent predilection, for, *mutatis mutandis*, the same could be said of the cervical spine, from C.IV to C.VII.
2. Both levels form the most mobile parts of the spinal column.
3. Both levels are situated, not topographically, but in a physiological sense, opposite the intumescence of the myelum.
4. During ontogenetic development, the tissues at both levels are very actively involved in the building of the body (cranially and caudally terminal division of the neural crest, sprouting of the extremities, plexus formation, physiological and unphysiological variations in neurological innervation)
5. There are many morphological anomalies of the spine at both levels; hence, as far as the lumbo-sacral region is concerned, the expression "lumbo-sacral anarchy". Let us now consider only the lower lumbar level.
6. Several dimensions between the low lumbar vertebrae are smaller than those of the high lumbar vertebrae; *e.g.*, the intervertebral foramina, in part due to the facts that the posterior aspect of the L.V vertebral body is less high, the incisura vertebralis superior of S I therefore smaller, as also the angle between the spinal column and the sacrum.

7. In an appreciable number of patients the available spaces between the moving vertebral parts are constitutionally smaller still (*e.g.*, flat wide lumbo-sacral spinal canal, fanwise extension of the lumbo-sacral arches).

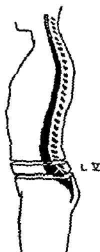
8. Let it be added that the contents of these relatively narrow spaces are the most voluminous in the lumbo-sacral region. Normally, the S.1 is the thickest spinal cord root, then follows L.5. The lumbar sac is often wide to pathologically dilated and is surrounded by a varying amount of fatty tissue.

9. The facts mentioned in points 3 to 5 have proved to form fertile soil for the occurrence of neurogenic anomalies in the lumbo-sacral region (thickening of the roots, anomalous splitting of the roots, ectopic ganglion).

All in all, these points show that, if only in a *spatial sense*, the lumbar-sacral region is a favoured locality for trouble, apart altogether from intervertebral discs and conditioning factors in the form of acquired changes.

The influence of the *erect posture* adopted by man is a difficult point to argue. Perhaps it will suffice to point to the distorted situation of L.V in relation to the fixed point of support, *i.e.*, the sacrum, which phylogenists regard as the result of this adoption of the erect posture.

The *greater weight* which the spine (especially the fourth and fifth lumbar discs) has to carry is accounted by some as the reason why disturbances are more liable to occur at the lower lumbar levels than elsewhere. Is this not pushing the argument too far?



At a rough estimate, a normally built individual weighs 2/3 X kilograms from the head to the level of the L.III vertebra, inclusive. The whole segment of the body at the level of the L.IV vertebra (roughly 5 cm) weighs some kilograms Is it likely that these few extra kilograms come to so much greater weight as to raise the chances of a disc lesion at L.IV – L.V by $\frac{46\%}{3\%}$? And why should not these chances in the case of the L.V – S.I disc – upon which the extra weight of a body segment again comes to bear – far exceed $\frac{39\%}{3\%}$?

In our opinion the external conditions for this predilection are as follows:

a. The lowest segment of the lumbar spine, immediately above the sacrum-pelvis, which is the fixed point of support, has to bear the brunt of *movements*. Although the amplitude of a movement is distributed as much as possible between all co-operating mobile segments of the spine, the lowest lumbar ones are hampered through being fixed at one side. This

implies that, whereas the necessary mobility is achieved at higher levels by the co-operation of neighbouring mobile segments, the lowest lumbar mobile segments have to act largely unaided. As a result, the normal course of a movement either involves the greatest local amplitude of movement in the low lumbar region (greater wear and tear), or inhibition of the necessary greater amplitude by the adjacent fixed point of support; and this causes torsion and, therefore, increased abrasion and degeneration of tissue.

b. Every movement involves the action of forces. The mass of the moving body, whether or not increased by the mass of a lifted object, is experienced at the other end of the "lever", the back, in the ratio of "force × force arm = load × load arm". As Armstrong (p. 32) and others have calculated, in stooping man the force arm is 15 times that of the load arm (and an object therefore "weighs" 15 times heavier in the back than on the hand). To understand the predilection for the place where that force acts, it is far more important to know that the force increases most – and therefore acts most powerfully – on the place where the force arm is longest, than to know the absolute increase in force through leverage. Now, even without full flexion or stooping and irrespective of the coefficient of multiplication, the greatest forces will come to bear exactly in front of the point of stabilisation, *i.e.*, the sacrum, in all movements of the lumbar spine. Therefore, through leverage, those forces which have been multiplied most will constantly cause torsion of the mobile segments L.IV – L.V and L.V – S.I, which are already the most inhibited in their movement.

This can be illustrated in a firmly fixed lath. Under rhythmic movements with increasing amplitude, the lath will break just above the place of fixture, the vibrations causing a predilection for this particular place. If the lath is pushed aside with the flat of the hand in one simple movement, breakage will occur at the same place, because it is there that the force arm is longest. A sudden blow on top of the lath, which causes it to bend at half its length, breaks it elsewhere. This may be compared to the effect of a single severe injury which flexes the spine forcibly, usually causing a fracture in the thoraco-lumbar region. Another clear indication is provided by another little experiment. If the lath is incised, it breaks at this site of least resistance.

With this in mind, we now have to consider the *conditional factors*, where the spine is concerned. Every factor referred to, with a predilection for the lumbosacral section of the spine, increases the probability of wear and degeneration at the site (joint, ligament, disc). The vicious circle is now closed; local wear and degenerative processes represent a new predilection for other secondary processes. Under the *precipitating influences* of movement and strain on the lowest lumbar segments, diffuse clinical pictures, to which the whole spine is pre-

disposed (osteochondrosis, osteoporosis, spondylarthrosis deformans, endogenously determined allergies), will manifest first and predominantly at these levels and, in their turn, will act as a conditioned place of least resistance in this localisation.

In point of fact, the local predisposition to one of the above clinical pictures, more especially degeneration of the disc and arthrosis, was found not to be entirely determined by the locally violent effect of wrong movements and strain.



Sphenoid vertebra — cleft vertebra.
Kissing spines.
Wide vertebral canal.
Spina bifida occulta of L.V and S.I.
Degenerated disc.
Spondylolysis — spondylolisthesis
(*open sacral hiatus, not visible*).



Trophic disturbances
at one or two levels.

The frequent coincidence of neurogenic anomalies with the morphological manifestations of a degenerative back, so called, together with degeneration of the disc at one and the same level, is indicative of (total) *trophesy* in that segment. We have noticed this coincidence repeatedly at a low lumbar level and there are no reasons for supposing that the same thing occurs to the same extent in the higher lumbar and thoracic regions. That is why we conjecture that there is a *constitutionally determined* predilection for degeneration of the

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suggested in the section on myelography, which we recapitulate briefly:

1. The indication for extensive examination rests predominantly upon the patient's clinical history and the failure of conservative treatment.

2. A correct assessment of the patient's psyche will at the same time show whether further examination and a more active therapy, or different treatment, are necessary.

3. In the course of further examination the results of fluid analysis (protein content higher than 50‰) and of the tentative radiography (*e.g.*, transitional vertebrae) will provide special indications for myelography. If those indications are not forthcoming, then careful neurological examination should be able to locate a possible disorder roughly at a level between L.IV and S.II. If there is positive evidence of a lesion of one (or more than one) root (L.5, S.1 or S.2) at this stage, exploration can be undertaken of the two lowest lumbar levels, this being extended along root S.2 if the clinical data specially suggest that course.

In mentioning some typical characteristics of disorders at certain levels, we wish it emphatically to be understood that the following syndromes are by no means complete. We are not mentioning minor variations and aspects, convinced as we personally are of their insignificance in practice, and submit only those typical facets which enable the examiner to differentiate roughly between a high lumbar and a low lumbar (or lumbo-sacral) disorder.

Typical characteristics for lesions at a high lumbar level (L.I – L.II and L.II – L.III)

(a) Prolonged clinical history, especially after an injury (at sport) in the past. The clinical history may record complaints resembling those of neuralgia paraesthetica. The first signs of the syndrome are sometimes betrayed by pain in the lateral aspect of the upper part of the legs.

(b) Acute exacerbation of diffuse radiating pain, especially in the thighs.

(c) These exacerbations are often accompanied by micturition, libido and potency disturbances, of which there are seldom any complaints in the early part of the history.

(d) After exacerbation, there may be residual diffuse paresis of the thigh muscles, but also of the peronaei on both sides. There is weakness of the quadriceps as well, manifested by the impossibility of tightening the patella.

(e) When exacerbation is acute, sensation is often temporarily disturbed in unconnected fields scattered over the whole leg, but more generally in the lateral and anterior parts of the thighs and in the lateral part of the calf.

(f) In the radiographs, local spondylosis deformans is seen on the anterior aspect of the vertebral bodies around the disc involved (L.I – L.II or L.II –

fourth and fifth lumbar discs. Viewed within the general framework of precipitating influences (wrong movements and strain), it will be clear that a dystrophic disc will be the first to come under the impact of these influences and to be most seriously deranged by them.

It appears, moreover, that when a morbid change has ultimately taken place at the lumbo-sacral level (hernia, affected joint, posterior lipping, etc.) as the result of a combination of many factors, the picture is further complicated by the coincidence that, just at this level, the relation between the available spaces and their contents is an unfavourable one, *viz.*,

Thick S.1 and L.5 roots.
Small foramina.
Flat spinal canal.
Large hernias (the lumbar nucleus pulposus being round and big, Schmorl, p. 16).

This relationship, which is determined constitutionally, is often particularly unfortunate, in that factors are involved predisposing to the limitation of space, *viz.*,

Flat wide spinal canal.
Transitional vertebrae.
Congenitally narrowed L.V – S.I intervertebral disc.
[illegible]

Hence it is due to a concurrence of many constitutionally determined factors that the favoured site for backache is the lower lumbar region.

Are "disc syndrome" and "root syndrome" correct terms?

There are objections to both these terms. "*Disc syndrome*" automatically implies hernia of the intervertebral disc mentioned, but what the clinician really means is "Complaints and symptoms, from whatever cause, at the *level* of the disc mentioned". "*Root syndrome*" is a theoretical concept representing a monoradicular lesion. In medical practice it conveys a topographical indication, *e.g.*, the "S 1 syndrome" implies that there is probably a hernia of the L.V – S.I disc as well. Level diagnosis based on the root syndrome is unreliable from individual to individual, on account of morphological and functional variations in the roots and unforeseeable special aspects of the spine and of the causal lesion

To what are we then to turn, since neither neurological nor radiological-myelographic diagnosis appears to provide any certainty? A rough guide was

negative on both sides and classical neural loss symptoms are often lacking in both lower legs. Irritation of or interference with the L.4 root is manifested by radiating pain, or sensory disturbances, in the tibia, sometimes radiating to the medial ankle.

(c) The knee reflex is sometimes found to have diminished or to be negative.

(d) Weakness of the peroneal muscles might be found.

(e) A local arthrosis deformans is sometimes found radiologically around L.III – L.IV.

In our material the L.III – L.IV lesion was seen quite frequently in combination with transitional tendencies of the spine.

Note

Why, it may be asked, if the fourth or fifth lumbar disc is so clearly the favoured site for abnormal conditions, do lesions nevertheless occur in certain cases at the level of the L.III – L.IV disc? The brief answer is that it is due to a a migration to that level of factors which, in other spines, have a preference for the L.IV – L.V and L.V – S.I levels. In other words, to the extent in which a given function of the spine and the local pressure of the spine have anything to do with this preference, we believe that in some spines the L.III – L.IV level constitutionally or conditionally does the work of the L.IV – L.V level. This was clearly apparent in our material for, in 75 % of the hernias at L.III – L.IV, we found a transitional tendency in a sacral direction. The hernias in spines with a transitional tendency in the lumbar direction proved to be localised at L.V – L.VI in 75 %.

– L.V
– L.V

These two sets of figures are in some way contradictory. In Krayenbuhl's sacralisation data, the anatomical L.IV – L.V level stood for the clinical L.V – S.I level, for the L.V vertebra was fixed by sacralisation, as vertebra S.I normally is. In our series, the anatomical L.III – L.IV level took, in the total function of the spine with sacralising tendencies, the place of the L.IV – L.V level of a spine without transitional tendencies and had the same predisposition to abnormalities as exists for a L.IV – L.V level under normal circumstances.

Literature on the L.III – L.IV syndrome. Bradford and Spurling (p. 79)
Zander and Brussatis (pp. 64 and 90)
Reischauer (pp. 6 and 47).

The typical characteristics of a radicular syndrome of the L.5, S.1 or S.2 root

It will be useful first to repeat briefly the course of the roots in relation to the disc and foramina

L.III), which need not necessarily have narrowed. It is not unusual to find a high protein content, e.g., 5‰ (normally 0.30‰) upon lumbar puncture.

Note

1. A syndrome of this kind – sometimes called a "transverse lesion of the cauda" in grave cases – is thought to be caused by the compression of several roots at the same time. The high lumbar canal is comparatively wide, but at this level it is filled up with many roots. The chronic nature of the neural loss symptoms is held to be due to vascular disturbances of the vasa radiculorum. Owing to trophic disturbance of the roots involved, the more weakly laid down innervation of the peroneal muscles falls away.

2. It is pointed out that a high lumbar hernia may produce syndromes of the low lumbar and sacral roots, which pass this level. However, it should not be forgotten that a disc lesion at a high lumbar level is itself liable to disorganise the movement of the low lumbar spine. If this disorganisation is maintained chronically, it is certainly well to bear in mind that the L.5 and S.1 roots, for instance, may be compressed in their own foramen and the corresponding lateral recess. This will occur all the more readily if a disc lesion of L.I – L.II or L.II – L.III causes a functional listhesis at a lower level. When operating in a high lumbar region, it would not be possible to deal at the same time with the complications at a low lumbar level. At first, a complicating functional listhesis will have to be treated conservatively (exercises or corset); then, if low lumbar symptoms worsen through, say, a secondary pseudo-spondylolisthesis, it will be necessary to consider a local intervention in the low lumbar region, possibly with an overhaul or fusion operation.

3. We know of cases in which the syndrome of a L.II – L.III hernia consisted only of chronic lumbar backache, (high) lumbar locking and an indication of lumbar scoliosis. The hernia was disclosed by suboccipital myelography, sometimes after lumbar myelography with negative findings, and finally proved at operation.

Syndrome of the L.III – L.IV level (root L.4)

Owing to its intermediate position between high lumbar and low lumbar/high sacral, the syndrome of the L.III – L.IV level presents certain peculiar difficulties. All the more valuable is it, therefore, to be able to recognise it by some rough yardstick. The more salient pointers to a L.III – L.IV syndrome are the following:

(a) In the clinical history, pain is intimated in the groins and lower abdomen.

(b) The neurological picture is poor in symptoms. The Lasègue tests may be

negative on both sides and classical neural loss symptoms are often lacking in both lower legs. Irritation of or interference with the L.4 root is manifested by radiating pain, or sensory disturbances, in the tibia, sometimes radiating to the medial ankle.

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Krayenbuhl (p. 39) noted the following coincidence:

12 sacralisations	10 hernia nuclei pulposi L.IV – L.V
5 hemisacralisations	5 hernia nuclei pulposi L.IV – L.V

These two sets of figures are in some way contradictory. In Krayenbuhl's sacralisation data, the anatomical L.IV – L.V level stood for the clinical L.V – S.I level, for the L.V vertebra was fixed by sacralisation, as vertebra S.I normally is. In our series, the anatomical L III – L.IV level took, in the total function of the spine with sacralising tendencies, the place of the L IV – L.V level of a spine without transitional tendencies and had the same predisposition to abnormalities as exists for a L IV – L.V level under [illegible]

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(a) In the clinical history, pain is intimated in the groins and lower abdomen.
(b) The neurological picture is poor in symptoms. The Lasègue tests may be

Sensory disturbances: (Hip) (lateral aspect of thigh, lateral aspect of lower leg), big toe.

Muscles: Weakened flexion or extension of the muscles of the big toe.

Root S.1

Radiation of pain: (Lateral aspect of leg below the knee), lateral edge of foot, little toe, heel.

Sensory disturbances: Ditto.

Muscles: —.

Root S.2

Radiation of pain: Posterior aspect of thigh and hollow of the knee.

Sensory disturbances: Posterior aspect of thigh, immediately above and below the hollow of the knee.

Muscles: Fibrillation of calf muscles.

Clinical history: Disturbances in micturition, diminished libido and potency.

Mixed syndromes = the complex of several different syndromes.

The many contributory factors involved in the production of these syndromes were enumerated during our discussion of the mechanisms responsible for low back pain.

It is customary in other branches of medicine as well to classify the whole clinical picture of the patient according to the dominant signs and symptoms. Thus a patient is sometimes referred to as a "gastric patient" even if suffering from-hypertension at the same time. Similarly, patients with back complaints are often classified as "hernia patient" or as a sufferer from arthrosis deformans. It is our personal opinion that such classifications seldom represent the true state of affairs. In the majority of cases the patient may well be afflicted with both hernia and arthrosis deformans. There are so many other factors to be considered, such as anomalies, actual traumatic residual conditions, infective conditions (?), etc. There will always be a mixture of constitutional factors, degenerative changes, exogenous influences and secondary symptoms. All these jointly form a mixed syndrome, which is further coloured by the patient's psyche, possibly with its own constitutional bias. Add to this that, whether or not constitutionally determined, the neurological picture is apt to vary, and the reason for the diversity of back patients' clinical pictures will be clear enough.

At the same time, the implications of this fact are important. Suppose a back diseased in its totality, as indicated above, suddenly develops exacerbations of one facet of the composite syndrome, say a radicular syndrome resulting from a hernia. The patient consults his medical adviser for *this* symptom and is, as a rule, treated for *it*. Further supposing that the surgical or conservative

The L.3 root passes the L.III – L IV foramen without approaching the L.III – L.IV disc.

The L.4 root passes the L IV – L.V foramen high up in the incisura vertebralis inferior. There is a slight chance of this root's running over the lateral part of the L.IV – L.V disc.

The L.5 root first passes the L IV – L.V disc, disappears into the L.V – S I foramen and then runs over the lateral part of the L.V – S.I disc.

The S.1 root passes the L.V – S.I disc in its course towards the intervertebral canal for the S.1 root.

Note

1. This topography, described in most of the relevant literature elsewhere, could be *said to be classical. But we should not forget that both the dural sac and the site of* emergence of the roots vary topographically in relation to the vertebrae and foramina. It is likewise known that the foramina, roots and discs vary topographically in relation to each other; hence the above "classical topography" could be modified by numerous exceptions. Add to this the many obvious anomalies in the morphological aspect of the vertebrae, the neurogenic tissue and its function, and it will be seen that the multiplicity of variation occurring in actual fact fully justifies the adoption of the term "lumbo-sacral anarchy".

The literature records Norlén's investigations, which were carried out on very much the same lines as ours, in the course of which we established many facts relating to the function of the caudal roots. With the patient under a local anaesthetic, the roots were mechanically stimulated after the anatomical relations of the spine had been correctly mapped out on the basis of plane radiographs taken of the whole spinal column. He comes to the following conclusion: "The extension and localisation of the sciatic pain can, in *certain* cases, disclose which root it is that *chiefly* directs the pain-impulses and can also give holds for the localisation of a disc herniation which may *possibly* exist". (The italics are the present author's.)

The doubtful reliability of topographical diagnosis, which is apparent from the careful wording of the quotation, is in close agreement with our own findings. In [illegible] e leg [illegible] ents, they intimated that the pain radiated to the lateral part of the leg.

In our opinion, those who seek guidance from a few typical characteristics of the monoradicular function of a given root are least likely to be misled if they bear the following in mind:

Root L.4

Radiation of pain: In the groin and low abdomen, possibly with extension along the shin to the medial ankle

Sensory disturbances: Along the shin, with extension to the medial ankle.

Muscles: (Weakness of adductors).

Root L.5

Radiation of pain· To the (hip), lateral aspect of leg below the knee, big toe.

of social disabilities and obstacles to the normal enjoyment of life. The only thing that matters to the patient himself is that he should recover his sense of well-being, and this is equally so if he is relieved of the distress *not* consequent upon a simple hernia.

It is our duty to weigh the chances of complete or partial recovery in every individual case, taking the nature of the disorder and prognosis of treatment into account. That is why it is so necessary to know something about the whole personality structure of the patient. It also follows, however, that the decision as to the treatment to be applied depends upon an examination of far more facets of the diseased back syndrome than merely that represented by hernia nuclei pulposi. These several facets have been given generally in the foregoing chapters and we recapitulate them here in the form of a chart.

A. Morphology	
Inferior muscular system	Asthenia.
Weak supporting tissue	Listhesis.
Dysplastic cartilage	Wear and tear of joints.
Dystrophic disc Disc degeneration tendency	Disc lesion.
Skeletal malformations	Relative stenosis.
Anomalies of nervous system and surrounding membranes	Dysfunction; limited space predisposing to stenosis.
B. Constitutionally determined variations in function of supporting tissue and nervous system	Strain, Dystrophy.
C. Constitutionally determined metabolic, allergic or involutional processes, including hormonal discrepancies	Spondylarthrosis deformans, diseases of the bone. "Conditional" de-adaptation in relation to the outside world.
D. Endogenously determined weakness of the vasomotor innervation and regulation	Physical incapability of sustained effort.
E. Disturbed (psycho) motoricity, constitutionally determined	Predisposition to sustaining injuries; strain.
F. Constitutionally determined psychic and social weakness of adjustment, partly manifested by neurotic and psychopathic forms of behaviour	Aggravation; disproportionate number of grievances. Mental incapability of sustaining physical exertion.
Constitutionally determined weakness of temperament	Neglect of counter-measures.
Poor intelligence	Failure to avoid precipitating factors.

treatment quells this severe exacerbation, the back nevertheless continues to be diseased in its totality, perhaps even more so from the after-effects of the necessary laminectomy.

Both the patient and many a clinician appear to forget these consequences. Over and above this primary treatment, the patient's whole condition has to be taken into account; it may entail surgical measures, such as providing more spare room, or the simultaneous elimination of other predisposing conditions to future trouble. After-treatment should be directed towards the recovery of the patient *in toto* and the restoration of a sense of well-being. Naturally, this calls primarily for physiological methods, such as movement and exercises, and not for those conservative expedients which enforce unphysiological conditions upon him (like rigidity and restriction of movement by corsets, etc.). For these reasons it should be brought to the patient's notice that his particular back is one which conditioned, or admitted of, this acute exacerbation before it occurred. He should be properly advised as to how to cultivate his back; he has, in fact, to learn to live (*i.e.*, to deal) with *his* particular back. Helped in this way, a great many patients, even those of middle age, will be capable of re-adjusting themselves to the demands of the outside world.

SUMMARY OF THE SYMPTOMATOLOGY

The symptomatology of the degenerative back

In this chapter we have discussed the many aspects of back complaints, with and without the complication of radicular symptoms. At every point the general symptomatology was compared with the unspecific syndrome of hernia, which has come progressively into the foreground. It is now held, erroneously and often to the detriment of the patient, that, not only is hernia most common in practice but that it is almost the only cause of backache calling for surgical treatment. In conformity with this view, diagnosis was bent entirely in the past on proving the existence or non-existence of hernia nuclei pulposi. Thus sufferers from radicular symptoms and backache were for all practical purposes divided into two classes, *viz.*, those qualifying for causal treatment and those for whom palliative therapy – often *ut aliquid fieret* – was prescribed.

In the chapter on treatment we repudiate the application of this rigid categorisation. Personally, we do not think there is any such dividing-line. Strictly speaking, it is not so much the hernia from which we relieve the patient as the syndrome of complaints and symptoms from which he is suffering. It is no less our aim to ward off threatened invalidism and thus to prevent a host

CHAPTER 7

THE DEGENERATIVE BACK AS AN ASPECT OF THE DEGENERATIVE HUMAN BEING

Pathogenesis of back complaints

Having discussed the mechanisms involved in the signs and symptoms of diseases of the back, we shall now enquire into the cause of these mechanisms and seek to discover the reasons why they occur in certain individuals. Is the aetiology of the diseased back known? What is the pathogenesis of that state of being which causes failure of adjustment in certain persons?

The following points should be considered:

1. The *incidence* of manifested degenerative backs. Is it so high as to form an integral part of back diseases as a whole? To our mind it is virtually impossible to establish the frequency with which people suffer from back disorders. Owing to the variety of signs and symptoms presented, the sufferer may have himself treated first by one doctor and then by another, or else not consult a medical adviser at all. All we know is that many people suffer periodically from back trouble, with or without radicular symptoms. Hirsch (see Spiegel 1957, p. 332), for example, reports that "65% of the population of Sweden have low back pain". According to Brocher (p. 28), the lumbago and sciatica morbidity of insured persons between 18 and 65 amounts to 4%, sciatica morbidity being 2.7% These figures should be compared with the morbidity of tuberculosis, *viz.*, 0.53%, established under the same conditions. McRae (see Spiegel 1957, pp. 308 and 327) suggests after a post mortem investigation that "nearly everybody 40 years of age or older has at least one posterior cervical and one posterior lumbar disc protrusion". It should be added that such disc lesions need not necessarily be associated with symptoms, or at all events need not produce the complete syndrome of a hernia nuclei pulposi.

We have tried to ascertain the incidence of the degenerative back in one or several of its aspects in the population, but could do no more than obtain a general impression. With the material at our disposal – neurological and neurosurgical cases – that kind of enquiry is soon brought up short by the difficulty of deciding which aspects are characteristic and which are not. We came to the conclusion that, in this material, the degenerative back was manifested in

When we speak of a "*degenerative back*", we mean the sum total of the constitutional manifestations predisposing man to spinal disorders, with or without radicular symptoms.

1. The signs and symptoms of the degenerative back may resemble in every particular the clinical neurological picture of hernia and arthrosis deformans, even in the absence of these lesions.

2. The syndrome of the degenerative back is induced by dysfunction of the supporting frame and motor apparatus and by consequent secondary changes, if any, the latter resulting from the unstable genotypical constitution, on the one hand, and disruptive exogenous influences, on the other. Through their interaction comes failure of adjustment or acquired de-adaptation.

3. The constitutional predisposition to failure of adjustment – from which few such subjects will be able to escape – is, fundamentally, a characteristic of a certain total structure of personality.

4. A personality structure of that kind *is* a certain "state of being". The degenerative back is a special aspect of this state of being.

We therefore say that a patient has a degenerative back if we recognise that he or she is not wholly adjusted, psychically and physically, and/or is to some extent de-adapted. Failure of adaptation may be betrayed by persistent signs of degeneration, also by a more or less pronounced psychic and somatic instability varying from individual to individual, with maladjusted psychic and somatic behaviour as the result. This maladjusted somatic behaviour finds expression in – thus endogenously determined – functional disturbances of tissues and organs, more particularly of the spinal column, supporting tissue and nervous system.

Strictly speaking, maladjustment, as meant in the context of the degenerative back, implies inadequate adaptation to the demands imposed. Further differentiation is possible as follows:

A. *Non-existent adaptation from the outset*, as when the neural tube does not close in the foetal stage.

B. *Adaptive weakness*, as when the young individual fails in proper adjustment.

C. *De-adaptation*, the word we have chosen to describe the condition of an individual whose constitutional potentialities are just adequate, so that in *youth* he appears to be adapted, whereas later on his adaptive capacity is insufficient to meet the, possibly heavier, demands of an altered bodily condition.

It will be evident from these three points that the age of the patient is liable to play an important role in this process.

The first failed attempts at adaptation may tell at an *early age*, as in the asthenic young girl who is growing too fast at puberty; she is saddled with a painful, sagging back. We suggest that possibly the "growing pains" of the child transiently de-adapted in his body-plan during growth belong to this category.

The *young adult*, the tissues of his body still young and his energy still unabated, stands up to external demands as best he can, calling upon natural reserves for effortless adaptation. It is only when the demands put too great a strain upon the disposition that external precipitating factors will gain the upper hand at this age.

Man ages in the whole of his physical and mental make-up. *Between the ages of 30 and 40*, the natural suppleness of the tissues begins to wane; movements become stiffer and excessive movements rarer; extra amplitude and more than physiological strain are avoided; adepts at their work, no matter how hard it may be, do not exert themselves more than is necessary; the body never again becomes as flexible as it was. Meanwhile, enslavement to the daily grind leaves little time for regular physical exercises; a person of this age lacks the desire and the energy to make them part of his daily routine. A bicycle ride for the sake of exercise, a swim to keep the body fit, are activities for which he has little stomach. Perhaps this is partly due to the fact that neither men nor women at this age feel so strong an urge to compete with persons of their own sex in the matter of physical condition, though there are, of course, exceptions. Possibly this urge is suppressed by the leading of, and becoming accustomed to, an uneventful existence with regular satisfaction of the sexual appetite. Maybe the competitive spirit does not find physical expression in this period of maturity, but seeks compensation in some other way, such as spiritually or in material outlets.

clear-cut forms in 7% and was apparent in subclinical forms in about 25%. The latter category consisted of patients in whom one at least of the signs was clearly present.

2. Are weak backs in all their manifestations *more prevalent today?* Nowadays this question is generally answered in the affirmative, though sound arguments in support of the contention are not advanced. We think it is difficult to answer, because of the various factors involved, *viz.*,

a. Public welfare being now far better organised, cases which would not have been treated formerly do now come to the clinician's notice.

b. This applies more especially to back disorders in so far as it is true that the "slipped disc" is a fashionable disease. That being so, not only will its diagnosis more readily occur to the practitioner, but the patient, having likewise heard of it, will be more inclined to have himself examined for complaints which were formerly ignored or neglected.

c. Again, thanks to Social Welfare, patients who formerly could ill afford to do so can now have themselves medically treated, without fear of the cost or of losing their jobs. This argument is so persuasive in its practical effect that *one sometimes wonders whether this social security does not encourage undue* preoccupation with backache.

d. Any comparison with former times would have to be based on records of back ailments, which, to our mind are not available as a reliable foundation. The incidental mention of back disorders or sciatic pain in older documents is by no means a touchstone for the frequency with which such ailments occurred. Furthermore, a comparison between recent medical literature and these records, even of a few centuries ago, reveals little from an anthropogenetic point of view.

e. Even if any indication were found in the oldest documents, it would be necessary to bear in mind that the average expectation of life two thousand to three thousand years ago was not much above thirty (Liselotte Bucheim, p. 108, Cartier, Kern). In the Graeco-Roman period, half the people died before they were 25 and as late as 1750 the expectation of life was under 30.

The vast majority of sufferers from back disorders at the present time are above 30 years of age. Apparently, people living 2,000 to 3,000 years ago showed the effects of great wear and tear, despite their youth, as we know from the work of Buchheim, who detected signs of arthrosis deformans, amongst other things, in the spines of Egyptian mummies.

3. Are we to consider a state of being predisposing to de-adaptation as a "premature" *ageing process?*

will now break down and be followed by de-adaptation. Such an extraneous circumstance may appear to be harmless; *e.g.*, spring-cleaning, lifting the same wash-tub moved countless times before, but this time, perhaps, filled a little fuller; a fairly good show put up at some sport in untrained condition; a minor trauma or comparable unco-ordinated, unpremeditated, sudden movement causing local strain. An attack of coughing, turning over in bed or putting on a shoe may be pathogenic to a back thus pre-formed, though let it be added that as a rule a pre-formed condition of the kind is likely to be constitutionally determined *in toto* as regards the time at which and the extent to which it prevails. In the ultimate, this predisposition is the syndrome of the degenerative back, while the disorders themselves (see chart, p. 515) are merely the various facets which this syndrome presents in all its variety as to timing and severity from individual to individual.

There are some people in *later middle-age* who do not seem to have passed through the consequences of this natural process of life. Yet, when questioned, many will recall having experienced this and that, transiently or otherwise, which they had passed off contemptuously as stiffness, advancing age, "rheumatism" and accepted as being some of those "ills flesh is heir to" when youth is past. Equivalents or other names for these vaguely-defined complaints are stiff back, sciatica, lumbago. Thanks to these people's psychical and physical "grit", however, their powers of adjustment have remained intact. And this is superlatively so in those exceptional cases of the elderly with backs entirely unscathed. Adaptation has a chance to proceed undisturbed and physiologically if the demands made upon the body are sensibly toned down to keep pace with the declining condition of the bradytrophic tissues and the muscular system. People in this category lead a calm life, have no pronounced osteoporosis, no arthritic diathesis, no degenerative stigmata, nor is the structure of their spine constitutionally predisposed to stenosis.

In broad outline, the condition (state of being) of the ageing human being is similarly described by Chapchal (p. 61), Lewin (p. 255), Milton (p. 61) and Sleewijk (p. 594).

The above sketch shows the influence of ageing upon evolving adaptation, *i.e.*, the condition. Obviously, breakdown processes can never influence the constitution, far rather may ageing (possibly premature through constitutional factors) of the whole being, or of one of the organs, be one of the predisposing agents causing back disorders at a certain age.

Note

Thus the ageing process has its say in various ways, *viz.*,

The condition of the body deteriorates, both as a natural course and from lack of physical culture. If a local predisposition already existed in the form of dystrophia of the bradytrophic tissues and if, from the start, the individual had little sense of body culture, then the constitutional and conditioned degeneration of tissues will progress by leaps and bounds. And if this degeneration strikes at the disc (which should not yet be sclerotic at this age), protrusion of the still motile, mucoid nucleus as a hernia is more than likely to ensue, whereas later in life and under different conditions this degeneration would have followed a more merciful course, taking the form of a disc lesion with spondylosis.

As a rule, the *fourth decade* is characterised by the more marked occurrence of breakdown processes, varying in degree from individual to individual and in period. We have explained what is meant by "premature involution" and when this can be said to prevail. In this period of their lives, those who do not take special steps to adapt themselves "become two years older every year", psychically as well. It is the age when one begins to get into a rut, of giving way to feeling older, the age of disillusionment, of resignation to the downward trend This ageing need not always be physically apparent; quite often it shows itself suddenly after some precipitating event, such as a serious illness, or a period of sorrow or anxiety. Then premature greying and an elderly skin mark the beginnings of the change in the individual. The teeth have often to be attended to, or have already been replaced by dentures; in women the menopause is ushered in early and disruptively. Thorough examination then often shows osteoporosis, paradentopathy, spondylarthrosis, incipient hypertension and so forth. There are already signs of corpulence; the patient himself or herself will admit that this has been coming on in the last few years since stiffness of the limbs has made them reluctant to indulge in bodily exercise. Not infrequently, the patient's way of life has changed just before or during this period, *e.g.*, he may take up a physically less strenuous job. Psychic changes of a varying nature likewise take place, when the ground-tone of the temperament dominates increasingly and the individual makes the impression of being predominantly depressive, impatient or querulous. Other psychic symptoms are more in the nature of compensatory reactions to this awareness of what seems an inevitable decline; *e g.*, brief spurts of energy, leading to determined plans to start a new life, often with social shipwreck as the result. Thus we have the morphological premature ageing process in the organs and tissues, the neglect of adaptive mechanisms and resignation to the physically manifested downward trend, side by side and activating each other. Should some external factor now intervene, the adjustment built up and maintained over the years

5. It is a known fact that man has added from 6 to 10 cm to his *average height* in the past 100 years (Günther, Schulte (p. 320) and Zaayer (p. 3599)). The latter writer assumes that the incongruity between the growth of the spinal cord and of the body (the so-called ascensus medullae) is further accentuated by this, with low back pain and radicular symptoms as the result. We are not armed with sufficient facts to contest this hypothesis, but we should like to point out that people of moderate height also suffer from back disorders. Increased body length is attributed to positive factors, such as improved nutrition. It is difficult to see how a better condition – in this case approximation to optimum nourishment – could predispose to degeneration of tissue or general degeneration. The disproportionate growth of a given person to a slim, tall, asthenic habit may, in individual cases, predispose the back to weakness, but to our way of thinking the emphasis then falls on the asthenia, not on the length of the body.

6. Many authors (Armstrong, Clarke, Lewin) associate man's *erect posture* with low back pain, on the assumption that, in the long course of his evolution, man has not yet found a means of compensating for the consequences of adopting this posture. Some stress the greater pressure thus coming to bear upon the lower lumbar discs; others lay the blame upon the fixed point, *i.e.*, sacrum or pelvis. An argument advanced is that hernia nuclei pulposi does not occur in animals, except horses, cats and maybe a few others. But this does not mean to say that animals never have low back pain. An ageing horse, urged on with the whip, does not complain. One cannot help wondering how a human being would react if, in spite of low back pain, he were made to move by force. Incidents from the difficult period during the late war may suggest an answer to this question, with particular reference to those persons previously suffering from many ill-defined back complaints who nevertheless managed to do all sorts of unaccustomed work under the pressure of circumstances (forced labour imposed by the occupying authorities).

The assumption of the erect posture may admittedly have shifted some of the weight of the body to the lower part of the back and the lower extremities, but this means that the cervical spine is partly relieved of pressure. Yet brachialgia is by no means rare, in spite of man's erect posture, though not as frequent as low back pain. Perhaps this supports the argument that it is not only the weight of the body which is involved.

The cervical and lumbar parts of the spine are its most mobile segments; from both sites much nerve tissue runs to the extremities and it is at these two levels that anomalies occur.

a. Physiologically at the appropriate age.

b. Prematurely, this being determined endogenously. It may be combined with other changes, such as a disc lesion. By having previously (between the ages of 20 and 40) developed a hernia, such a patient stands out, as far as his back is concerned, from the large group of human beings ageing prematurely or at the physiological time, his back having already produced symptoms. We therefore have two large groups of persons above the age of 40 – taking this roughly as a line of demarcation, *viz*.,

In addition to the normal signs of ageing, one group is conditioned to a weak back on account of having developed a disc lesion, whether or not with herniation.

Roughly speaking, the second group has passed the time for herniation, probably on account of scleroticising disc tissue. The influence of premature or physiological ageing seldom produces a hernia in these people; it may lead to a mild disc lesion, but is usually confined to starting off the processes of wear and tear, without specifically affecting the disc.

4. Do we know of *exogenous factors* which favour the formation of the constitution we have in mind?

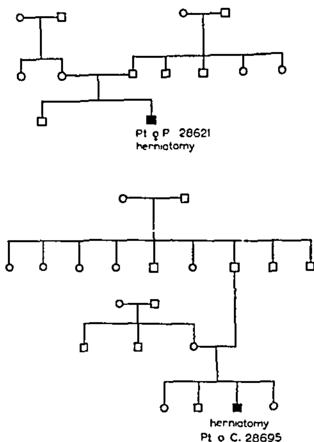
The fact that the constitution is determined endogenously would imply that in that case certain genes would have had to be influenced by exogenous factors. Little is known with certainty about this matter, *e.g.*, the effect of radiation. Actually, to assume it would be contradictory. Exogenous factors, on the other hand, are to be regarded as precipitating factors in the development of a physical condition.

a. As Biemond and Prick have demonstrated in laboratory animals, an intervertebral disc may degenerate through an infective disease of the spinal canal and may even herniate. In practice, no signs of inflammation are seen in the extirpated tissue of a diseased intervertebral disc.

b. Leaving rare cases (sarcoma of the vertebra, etc.) out of account, tumours are never found.

c. Disorganised calcium metabolism, protein metabolism or hormonal discrepancies are to be regarded as precipitating factors changing the condition of the spine.

d. Trauma in all varieties is an exogenous factor to be dealt with in another chapter. A trauma can never be the cause of a condition determined constitutionally. Let it be said here and now that, apart from exceptional circumstances with fractures established beyond doubt, we do not know of a single case in which the trauma could confidently be asserted to be the only cause of a disc lesion.



position to diseases of the back. Brocher, p. 30, comes to the same conclusion. For all that, our investigations produced reasons for believing that, in exceptional cases, powerfully precipitating exogenous factors were able to cause a hernia in the absence of a demonstrable hereditary predisposition. Lastly, we should like to say that it is the study of family histories that makes one especially aware of the diverse ways in which disorders of the back manifest themselves. To recapitulate:

Predisposition to diseases of the back is fairly prevalent.

Exogenous factors may precipitate diseases of the back, but have nothing to do with the endogenous predisposition to them.

The increasing average stature of the human race may tend to favour back disorders in certain individuals.

Although man's as yet incomplete adaptation to the erect posture may be a component of his constitutional predisposition, it cannot, to our mind, be responsible for the total regressive condition of the degenerative back.

It seems highly probable that disorders of the back run in families.

7. Is predisposition to a diseased back as a whole, or to one of its aspects, *hereditary?*

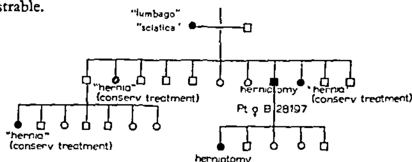
The literature has little to say on this subject. We know families in which this predisposition appears to run. Here are some examples:

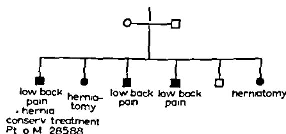
a. Twins had a hernia at the same period of life. (Prick and Hoeberechts.)

b. One of a pair of female twins began to suffer from low back pain, without demonstrable cause, at the age of 17; the other girl was watched anxiously, though of course without her knowing it. Six months later, exactly the same thing happened to her.

c. *Spondylarthrosis in the family. Between the ages of 33 and 35, three sisters* began to present the signs, confirmed by X-ray, associated with the same disabilities from which one of the parents had suffered.

d. The occurrence of hernia, low back pain, radicular symptoms (lumbago sciatica), either or not combined with signs of degeneration, in families is demonstrable.





There are few families in which these disorders or symptoms do not occur in combination. On the other hand, we know of some families in whose long history only one single case of hernia occurred. A few examples chosen from dozens of family histories are given on the next page.

We think there is some foundation for the acceptance of heritable predis-

The coincidence of a degenerative disorder of the disc and a degenerative stigma of the nervous system at the same level is striking, *e.g.*,

Anomaly of the root plus a disc lesion with or without hernia.
Syndactylism of 4th and 5th toes plus a lesion of L.V – S.I disc.
Transitional vertebrae plus a disc lesion with or without hernia.

There are also other frequent combinations at one and the same level, notably: spina bifida occulta – wide spinal canal – wide dural sac – anomaly of the root – disc lesion [2]).

The following cases are interesting.

Pat. Z. 291055. Klippel Feil's synostosis + meningioma at the same level.

Pat. B.N. 1254. Low back pain, radicular syndrome, neurosis, "rheumatism", uterus myomatosis, kyphoscoliosis, wide spinal canal, spina bifida, cauda meningioma in the thoracic spinal canal.

Pat. Bi. R 27483. Syringomyelia, polydactyli, trauma at L.II – L.III or residual condition of infection of a disc, Bang's disease, functional listhesis L.IV – L.V.

Re [1]) We encountered vasolability as a constituent of the syndrome fathered on Eppinger and Hess, *viz.*, "constitutional anomalies in the autonomic innervation" (Hoelen, p. 253). Von Bergmann speaks of "vegetative stigmatisation". The syndrome in these patients consists of vasomotor lability, of the action of the heart, blood pressure, glandular secretion and of the metabolism; further, peristaltic disturbances in the digestive tract and hypersensitivity to allergens.
Re [2]) From observations in practice it appears that the diversity of coincident somatic stigmata is immense, *viz.*,

In the skeleton: Spina bifida occulta, pseudo-spina bifida occulta of S.I – S.II, total dysrhaphia of the sacrum, rachischisis sacralis, transitional vertebrae, wide spinal canal; lysis of the arches, (pseudo)-listhesis, endogenous degeneration of the disc.

In the nervous system: Alternating roots, thickened root or lacking altogether, joint origin of two roots, anomalous transitional fibres; ectopic ganglia.

In the membranes: Cystic root, perineural cyst; meningeal diverticulum, "miscarried" meningocele, epidural cyst, sacral meningocele, meningocele (= patent spina bifida); wide cystic dural sac; shallow dural sac.

Combined morphological and functional anomalies of roots, ganglia and membranes

We are persuaded that these anomalies are to be seen as correlated, more or less pronounced manifestations of a single disturbance of development. It may be present in one organ or in several simultaneously and, as often happens, it may occur in conjunction with manifestations of a developmental disturbance,

Before we proceed, let us see what other facts are known, turning for some to the literature.

The syndromes of back disorders, with and without radicular symptoms, are also found unassociated with herniation of the disc (facet syndrome, abnormalities of the joints (Milton, p. 44), lipping (Bick, see Spiegel 1956, p. 175)).

Roeder (p. 11) points out that the coincidence of anomalies (transitional vertebrae, etc.) and hernia is not a mere matter of chance.

The combination of hernia and congenital anomalies occurs in families (Biemond, p. 233).

Von Muralt (p. 120) ascribes "sacralisation sciatica" to a coincident hernia.

Bauer says that if one component of the supporting tissue is badly laid down, the others also appear to be imperfect in varying degrees ("Mitbeteiligung anderer Mesenchymaler Gewebskomponenten"). See Boeters III, p. 107.

Kühne says that it is not a particular anomaly that is heritable, but the tendency towards the formation of anomalies (see Bauer, *ibid.*).

There is such a thing as a congenital inadequacy of the disc (Zaayer, p. 177).

Passages in a similar sense can be quoted from Curtius, e.g., "*Die organischen* und functionellen Erbkrankheiten des Nervensystems".

Sillevis Smitt wrote about "the morphological abundance of heredo-degenerative signs".

It is necessary to distinguish between acquired stiffness, owing, for instance, to a decrepit muscular system (as in the case of arthrosis deformans), and innate stiffness and awkwardness (Burger).

In the medical literature of the world, a constitutional predisposition to back disorders is accepted with conviction in the main, though here and there hesitantly. On *theoretical grounds* alone it would appear probable, for why, everyone must some time ask himself, should one person suffer from them and another not under (apparently) the same circumstances?

In *practice* we come face to face with Sillevis Smitt's "morphological abundance of heredo-degenerative signs" in sufferers from back complaints, *viz.*,

Degenerative signs		
Skeletal anomalies	With and without hernia	Low back pain
Anomalies of the nervous system		
Anomalies of adjacent membranes		
Anomalous functions		
Vasolability [1]		Radicular symptoms
Labile psycho-motoricity		
Labile psyche		

lodysplasia (*e.g.*, anomously transitional fibres). This recalls the oldest descriptions of the status dysrhaphicus in which it was typified as a combination of myelodysplasia and dysrhaphia.

Let us return to the pathogenesis of the predisposition to diseases of the back. In the preceding 1 to 7 points, there was only one dubious predisposing constitutional factor.

8. In view of the correlation suggested above, and shown to exist in practice, it is our personal opinion that there is a close relationship between a family constitution predisposing to back disorders and the status dysrhaphicus. In the literature, a repeatedly coincident phenomenon bearing typical dysrhaphic hall-marks is reckoned as a component of the status dysrhaphicus; we claim the same right to class the so-called degenerative back as a type of constitution. As has been said, there is no consensus of opinion in the literature as to which coincident phenomenon is in the status dysrhaphicus category. It likewise depends on the personal bias of the reader whether he identifies the degenerative back type of constitution with, regards it as a component of, or sees it only as remotely related with, the status dysrhaphicus. Perhaps it would be more correct to assign certain aspects of the status dysrhaphicus to the extensive type of degenerative constitution which predisposes to back disorders.

If, however, any connection whatever with the status dysrhaphicus is denied, then there is no alternative but to place the degenerative back in a class apart. In that event, there would be, mingled in their manifestations:

The degenerative back as a predisposing type of constitution;
the status dysrhaphicus as described by Bremer and Curtius; and
Sillevis Smitt's micro-heredo-degenerative myelodysplasia.

Collectively, these could be called: *a heredo-degenerative dysrhaphic constitution.*

Our arguments on the pathogenesis of back disorders, then, have led us to the point where a predisposing type of constitution is acceptable and the assumption of some relationship, whether close or remote, with the status dysrhaphicus and micro-heredo-degenerative myelodysplasia appears to be reasonable.

Of what fundamentally *causes* this constitution, we know nothing. All we do know is that the predisposition to back disorders is probably hereditary; and it has been shown that certain aspects are dominant, while others are recessive.

Let us, in conclusion, ask ourselves the following questions:

Is this heredo-degenerative dysrhaphic constitution (henceforth again to be called 'degenerative back') to be considered as a result of *cultural decadence?*

Bremond (p. 76) says that degeneration of the disc at a youthful age is a

tion in the skeleton. We know of "microdysplasias" – demonstrated by fibre counting and in other ways – from Verhaart's publications.

The frequent occurrence of *psychic disturbances* together with many manifestations of the status dysrhaphicus has been the subject of much study. Curtius, Bremer and Sillevis Smitt, as well as Bijl in his monograph of 1956, all consider this coincidence to be significant. Curtius and Lorenz (1934) investigated this closely. They mention the biological and at the same time social inferiority of a large number of persons examined, emphasizing the high incidence of psychopathy and neurosis.

Curtius adduced evidence of *dominant heredity*, pointing out that, although the tendency towards anomalies is heritable, the patterns in which this tendency is manifested may vary in different members of one family. He even calls it an exception for parents and children to bear the same degenerative stigma. Given this inherited disposition, he says, it may lie dormant until awakened or strengthened by external factors not yet known.

There are others who invest the status dysrhaphicus with a *pathoplastic* significance. It is said to aggravate the predisposition to infection and the consequences of injuries in the area of the dysontogenetic segments of spinal cord and vertebral column.

We established empirically the significant coincidence in back patients of:

a. Typical signs of the status dysrhaphicus.

b. A diversity of morphological anomalies in the nervous system and spine.

c. Anomalous functions of the nervous system, manifested as variations in the reflexes and innervation areas, etc.

d Psychic disturbances.

Re c. We have reported on these fully in the chapter on neurological disturbances. The encircling, band-like innervation areas, *e.g.*, around the lower part of the leg, described by Bremer and Curtius as a component of the status dysrhaphicus, are frequently seen The regularity with which they occur in persons of the same type of constitution argues in favour of the assumption that this variation springs from an organic basis (an anomalous arrangement of fibres around the central spinal canal, according to the above writers)

(If they should be regarded as psychogenic sensory disturbances, however, it follows, *ipso facto*, that the patients must be placed in category *d*, *viz.*, coincident psychic disturbances)

Re d. The psychic disturbances described in the relevant chapter strikingly resemble the quasi-neurotic and quasi-psychopathic conditions which Bremer, Curtius and others observed in sufferers from the status dysrhaphicus, also the mental instability noted by Bijl.

Re b. and c Morphological and functional anomalies are traceable in part to mye-

fittest. This rule was applied to all engaged in the struggle for life through all the ages. What will happen if this struggle for life becomes less exacting?

History records the physical prowess of which our ancestors were capable; the weight of the weapons they bore and their ability to withstand great hardship and privations testify to an optimum physical condition. Practice – maybe from pure necessity for the preservation of his life – can put a man into optimum condition, but that condition is outstanding only if the disposition is favourable. The hard life man led some thousands of years ago – in hunting, the use of arms and the constant fight against hunger (Cartier) – furnished the ground for that selection which depends on Darwin's principle of the survival of the fittest.

It would be short-sighted to see this principle in all its ruthlessness as applicable only to the life of primitive man; the saying 'homo homini lupus' has been true for all ages. The obvious conclusion is that the progeny of the fittest by natural selection was the most numerous; child mortality was held within bounds by strong protection and comparatively adequate nourishment of the family. The influence of this natural selection declined when culture softened the conditions of life.

The political and social events of our time are reciprocally related with the fact that social welfare, together with organised mechanization, keep even the mentally and physically unfit alive, for the simple reason that they exist. With the present trend of social security, there is no let to the number of progeny, nor, leaving manifest cases of degeneration out of account, is there any reason why such progeny should be less viable. Arguments can, on the contrary, be advanced (see, amongst others, J. H. van der Hoop, "Verspreide Geschriften", Arnhem, 1952) to show that artificial social security tends to increase the number of children in precisely that class of the population which, despite lack of constitutional soundness, is relieved of the necessity of itself providing for this security.

Among other things, the co-survival of the *un*fit has the following two consequences.

1. Individuals saddled with a dominant hereditary constitution, through which they would not be viable under less favourable conditions, are nevertheless kept alive and beget children. Not only do the bearers of such a constitution increase in numbers parallel with the growth of the population, but, within certain limits, those numbers increase as culture develops further.

2. This does not only apply to the morphological aspects of the constitution; it includes psycholability, psychogenic and neurogenic disturbance of locomotion, briefly the whole gamut of the still unknown dominant or recessive

cultural phenomenon. We should like to put it more broadly, *viz.*, the degenerative back type of constitution that predisposes to this degeneration of the disc *sui generis* is a decadent cultural phenomenon. Failing anthropological proof, reasonable arguments can be advanced in support of this hypothesis (Hanraets 1956).

Coincidence of state of culture and back disorders

In this context we mean by culture the influence of the complex of mental, spiritual, social and material factors by which the conditions of human life are directed from the natural state. We have no statistics showing the incidence of back disorders among primitive peoples (*e.g.*, Papuans). When we were in Indonesia (1948) we were struck by the comparative rarity of back disorders among the Indonesian population, in sharp contrast to their extensive prevalence in Europe. Neurologists who had worked for many years there, corroborated this impression. In the exceptional cases of hernia nuclei pulposi among Orientals, these always turned out to be of pure or mixed Chinese descent. The Chinese are one of the oldest cultured peoples of the East.

According to reports from America, which have been quoted earlier in this book, the incidence of back disorders in the white population is four times that among coloured people. There can be no doubt but that, as their historical development shows, certain coloured races are closer to human nature than the white race taken as a whole. It is interesting to recall that a complex of symptoms resembling low back pain and lumbago is observed in some domesticated animals, *e.g.*, horses, pedigree dogs, especially dachshunds.

Then, the population can be classified, not only in groups according to the cultural historical development of the race, but also according to individually varying standards of cultural development. The tenor of some lives is governed by the individual's constitution. Also, however, an accumulation of external factors (upbringing, social and cultural influences), interacting with this individual constitution, may build up a physical condition which, in actual fact, determines the person's tenor of life, either in a positive or negative sense. In some cases it is difficult or impossible to distinguish between the constitutional and conditioned attributes.

It is our impression that culture (evolution as from the natural state) not only sets limits to the physical individual condition, but has for centuries been directing the physical constitution in a negative sense.

The influence of culture on the constitution

Darwin's concept of evolution rests on the principle of the survival of the

fittest. This rule was applied to all engaged in the struggle for life through all the ages. What will happen if this struggle for life becomes less exacting?

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2. This does not only apply to the morphological aspects of the constitution; it includes psycholability, psychogenic and neurogenic disturbance of locomotion, briefly the whole gamut of the still unknown dominant or recessive

heritable manifestations of the degenerative back. The process began when *mental and somatic stability was no longer necessary to the preservation of* life; and it has been going on steadily ever since the optimum chances of life no longer depended on fitness. Nowadays, persons who are for ever stumbling, falling down, receiving minor injuries – in short, the clumsy ones – risk little beyond straining their backs, against the consequences of which they are, moreover, insured.

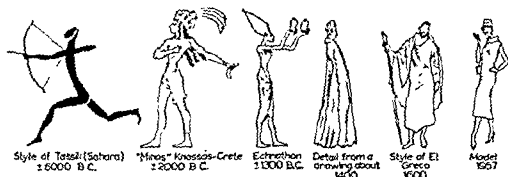
To illustrate this we quote from Sheldon's *Atlas of Men* (p. 250): "Possibly the survival of somatotype 5.1.4 (ectomorphic endomorphy at extreme mesopenia) at all as an adult man in a human society should be attributed to the operation of *cultural* rather than biological influences... The question is appropriate: Would 5.1.4's be found in a sample of population living under harder or sterner conditions?" Page 132: "Somatotype 3.1.6: the bony skeleton and the musculature... are just barely sufficient for the maintenance of human life, and it may be too that they are sufficient only under certain favorable conditions such as those currently prevailing in American Society. Possibly a... 3.1.6 cannot or does not exist as a male in parts of the world where more rigorous standards of survival are in force". "In the male this is a borderline somatotype, just viable. In the female it may in our culture be fairly well adapted".

The decadence through cultural influences described above is the result of less selection with regard to positive constitutional qualities. Hand in hand with this, selection has been taking place right down the ages in favour of those attributes which were bound to influence the constitutional strength of the frame adversely in the long run. We are referring to controlled breeding as has been proceeding, through culture, especially among the white races. What is going to happen if, for a prolonged period, poultry breeders only allow the eggs of hens gifted with a given quality to be hatched out?

It seems highly probable that controlled natural selection will be exercised by man in favour of slenderness of build. We know from archeological work that many thousands of years ago men were more stockily and strongly built than is the phenotype of today. Kern *(Historia Mundi)* puts this point in his contention that primitive man was completely human *in spite of his figure*. Cartier says that skeletons of human beings of the Middle Paleolithic give us some idea of their height and appearance. It is thought that their skin was black and that both sexes wore beards. The phenotype of European man at the beginning of our era is comparable with that of the whites of today, but the circumstances under which the subject classes then lived were still such as to require an optimum condition on a constitutional basis to "preserve (the) favoured (races)

in the struggle for life" (freely rendered from the sub-title of Darwin's "Origin of Species"). It was the women who did the heavy work and they had the physical build for it; indeed, they needed it for what they had to do and it was probably rather for their strength than their beauty that they were chosen by their mates, as is still sometimes the case, though more and more exceptionally. First Christianity and then the cultivation of courtly ideas in the age of Chivalry improved woman's status in a multitude of ways. As taste became more refined, so man became more fastidious in his choice of a partner.

A preference for slenderness is evident from the paintings and literature of the times. Artists idealise their subjects, giving them [illegible] gonists, male and female, are [illegible], in fashion plates – which, a[illegible] [illegible]s desired, rather than represent what is – the figures [illegible] elongated out of all proportion. History teaches us that this preference has existed right down the ages. There is a striking contrast between the realistically described appearance of Roman matrons and



the frail, ethereal figures portrayed at that time. The contrast in bodily build between the idealised Don Quixote and his slow-witted, genial attendant, as described by Cervantes, was certainly not devised by chance. The elongated, almost tapering figures in El Greco's pictures are familiar to all. Goya's caricatures of his rich clients show figures of inaesthetic proportions, but when he was free to choose his own subject, whether devotional or political, his aestheticism was undefiled. Van Eyck idealised the figure of the woman in his well-known painting called "Adam and Eve" to the extent of distorting the correct anatomical proportions; again, this figure is slender, yet Van Eyck had to pick his models from a population consisting largely of the stocky Celtic type. There are, of course, individual exceptions and their imitators; the expression "Rubens figure" corresponds to a representational manner and a preference which were the fashion in a certain period.

An enquiry reported by Sheldon (p. 89) bears out the foregoing. "In a study of about 150 paintings of the Christ, reviewed at the University of Chicago in 1938, it was found that 30 per cent. of the artists had pictured the Christian central figure as nearer to the 2.3.6 than to any other somatotype; and about 35 per cent. had seemed to portray Christ as most closely resembling the 2.3.5" (2.3.6 and 2.3.5 = delicate, tall, long-legged). "This may indicate only the persistence of artistic fashion and its reflection of a cultural ideal".

The artist's idealised wish-fulfilment interprets man's age-long preference for the slim type of woman. We think this can be psychologically explained by the fact that slenderness is identified in the human imagination with youth, elegance and beauty; moreover, the type of woman who appears to be fragile will appeal to the masculine urge to dominate.

Sheldon (p. 24) states the case upon which we base our hypothesis in the following words: "After adolescence, nature slims her children for the mating dance". Veraguth (p. 21) tells us that the response of his audience to a series of pictures shown expressed a decided preference for the tall, slim type.

Popular taste follows the lead of the governing class. In step with social development, the tendency towards refinement quickly spread to the lower classes of the population. The enfranchisement of women from slavery, the elaboration of court life, the formation of guilds and other organisations, as well as the emancipation of the common people, are all part of the evolution from the natural state of mankind. Through evolving culture, the product of natural selection resulted in a more refined type of woman and, *via* her, in a taller, slimmer race.

We may well ask whether the increased average length of Europeans, which is an established fact, is due to the more intensive application of natural selection described above – leaving other theories out of account, such as improved nutrition. It has swept beards from women's chins, given them a more delicate skin, an oval face and, on the whole, a less robust habit.

Buytendijk (1951) considers the phenotype of modern woman to be a cultural decadent build. Frets (1950) assures us that the Mendelian laws apply to the phenotype of the human, quoting de Vries and Morgan (the length of the body is a hereditary characteristic of the race), Davenport (the dimensions and relative proportions of parts of the body are hereditary); Frets, Kuhne, Fischer (hereditary variations of the vertebrae). Both Kretschmer and Schlegel showed that the asthenic and leptosomic habit is hereditary. It is therefore understandable in the light of Mendelian laws that natural selection in favour of the slender type generation after generation eventually led to a taller, slimmer race, *i.e.*, to a weakened constitution as far as the frame is concerned.

It may also be inferred that if strongly built men of many generations chose slender women for their wives, some of their progeny might well have the powerful build of the father, yet internally the weaker frame of the mother.

This natural selection is effective in two ways, *viz.*,

1. Quantitatively through selection of the privileged attributes (more offspring as the result of marriage at an earlier age and of enhanced libido due to having conquered the chosen mate).
2. Because it ultimately leads to a kind of in-breeding, which promotes the heredity of selected characters.

Summing up these arguments, we find that culture can influence:

1. The human *condition* of every individual.
2. The human *constitution*, *viz.*,

   a. by less selection in respect of positive characteristics – in accordance with the principle of the "co-survival of the unfit", owing to which dominant hereditary negative factors spread.

   b. By directed natural selection, which furthers in-breeding in respect of certain attributes, through which the recessive heritable factors may also ramify.

We now also have to ask ourselves whether exogenous factors capable of modifying the individual or the species are elicited by cultural influences, thinking more especially of changes to the gene pattern as the result of radiation.

The matter being beyond our competence, we quote from Ganguli's paper, which was lent to us by the Journal of the Indian Medical Association:

"According to the modern concept of evolution, all normal genes are mutant and the succession of mutations is the basis of evolution. In the history of mankind, spontaneous mutations have occurred and re-occurred over countless generations to give rise to what the present man is today – the result of survival of the fittest and natural selection. This spontaneous mutation rate increases when exposed to radiations and chemicals, but no new types of mutation occur. The increased rate of mutation will cause acceleration of evolution. ...Human beings are subjected to natural sources of radiation, *e.g.*, cosmic rays, and got themselves adjusted to the effects of this radiation in such a way that mutations occurred in a desirable and beneficial way. ...The creatures over the earth have always been exposed and will remain exposed to natural sources of ionising radiation. But the danger that looms large, is not due to the nature of the hazard but the ever increasing quantity and that is new. ...Far-reaching is the effect of radiation on the germ cells. Structural alterations of the chromosomes produce 'chromosome aberrations', Stone R.S.,

Radiology, 58 (1952) 639, Keosian J., *Science* 122 (1955) 586. The chromosome effect is dependent on the dose rate and acuteness of exposure requiring only a few roentgens. .... The effect of radiation on genes is additive. The accumulated dose and not its intensity or duration of application is responsible for the frequency of mutation. ... Radiation will cause acceleration of evolution. ... Based on the present day knowledge of genetics, evolution and biological phenomena, it will not be wrong to infer that blind mutational changes without regard to the end results due to unadapted additive radiations are not only not beneficial but detrimental for man. ... At present it is unlikely that *mutations* would cause a distinguishable effect in this generation or the next resulting in premature death, or failure to reproduce. Again, for each mutation drastic enough to be recognisable in the first or second generation, there will be hundreds of others with effects too small or too hidden to be seen. A man born with a damaged gene may have a slight defect, but if he marries a woman with a damaged gene, the child will show the defect more conspicuously. The handicap will not only go down the line of descent but also grow to be a major *permanent handicap in future generations....*

The biggest problem a medical man has to face is to estimate what amount of radiation may a person be exposed to without damage to himself or the future generation. ... At the present moment exposures from X-rays industrial or medical, of whatever power, may cause more genetic changes than a lifetime work with radiation (radio-isotopes etc. being used in laboratories and hospitals). ... It is therefore imperative that uncontrolled, unsound and ill planned installations of unnecessary sources of X-radiation whether experimental, diagnostic or therapeutic, must be abolished...".

Similar and other observations on this subject are to be found in recent literature (Lea, monograph; Aten, p. 748). Agreement does not appear to have been reached yet as to the critical dose, nor on the degree of danger to which man is exposed by radiation. To estimate these values by comparison with the amount of natural radiation to which human beings are exposed on earth with impunity seems to us a questionable procedure. For, what proof have we of this impunity? According to Muller and Fischer, the possibility of "chance" evolution without the action of selection can be ruled out. We read in Huxley (*Evolution as a Process*) that it is mainly the small mutations which make their mark on evolution. Evolutionary change is nearly always gradual and is brought about almost entirely by selection.

We cannot deny the possibility of an effect upon the genes of long ages of "natural" radiation, the result being a change in the human constitution *via* mutations and selection Parallel cultural influences are diminishing selection

in favour of positive qualities and increasing selection towards attributes with a negative tendency in a certain respect. Not inconceivably, products of generation (in this context, mutations as the result of natural radiation) were able to hold their own and reproduce their kind under the milder conditions brought about by culture, whereas this would not have been possible under the harder conditions prevailing in former times. Thus it may be a tenable proposition that an unknown proportion of humanity, living under the conditions produced by "culture", is endogenously endowed with constitutional attributes brought about by the influence of natural radiation, and that the, maybe comparatively higher, incidence of those attributes today is perhaps due to natural radiation (mutation) *and* the effects of culture.

It will be evident that we count the heredo-dysrhaphic constitutional type of the degenerative back among these attributes. If this is so, and assuming that in certain cultural fields the dose of ionising rays for medical, industrial and other purposes is just as large for the whole population as that of natural radiation (though also taking into consideration that so-called cultural influences tend rather to increase than to diminish), we may expect this type of constitution to increase progressively both in number and in clearly marked manifestations *.

Our arguments for calling this heredo-dysrhaphic constitution *degenerative* and for speaking of the "degenerative back" are the following, with reference to the definition of the concept, degeneration, given on page 162.

Every descending process of development may fairly be called degenerative, the meaning of the word then being indeterminate and approximately as it is used in belles-lettres. The descending trend is clearly evident from the presumed weakness, predisposition to illness, the influence of a decadent culture. In the total picture of the condition outlined, the negative character of this constitution, as it has come to be, is constantly manifested by disturbances; seldom or never by a positive quality.

Those who have this constitution bear typical stigmata, which in medical parlance are termed degenerative.

* "*The Hazards to Man of Nuclear and Allied Radiation*" (report) London (1956): (p. 65, Risk by X-ray examination of the Lumbar Spine); "*The Biological Effects of Atomic Radiation*" (report), Washington 1956; Spaander (p. 3110) "Over verhoging van dominante en recessieve erfelijkheid door radiatie" ("On increase in dominant and recessive heredity through radiation"); Van de Vooren (p. 1647), "Over het verband tussen de mutatie frekwentie (door radiatie) en vruchtbaarheidscoefficient (cultuurinvloeden)" ("On the relation between the frequecy of mutation (through radiation) and the coefficient of fertility (cultural influences)").

Apart from these general considerations, there would seem to be good reasons for regarding the dysrhaphic factor, more particularly, in this constitution as degenerative. We turn to a study by Jelgersma (1955) for further clarification. Commenting on his comparative anatomical work, he makes the following points:

I. "*Dysrhaphia occurs (more) frequently (than was hitherto suspected)*"

Dysrhaphia occurs so frequently that the aspects mentioned are no longer regarded as an abnormality, but as a characteristic feature of a variety in the species. This contention is upheld by several arguments, which we can support from our own experience. Some skeletons in anatomical collections proved to be dysrhaphic, though the fact had not been known previously. Far more dysrhaphic features are recognisable in textbook illustrations than are described there.

II. "*The more natural selection there is*," (read: in-breeding through selection) "*the greater the incidence of dysrhaphia*".

This can be verified in the descendants of cross-bred sub-species of dogs and those of pedigree animals which are the product of strictly controlled breeding.

III. "*The dysrhaphic human being is an atavistic variety of the mammal*".

In order to appreciate the implications of this statement, it is necessary to recall some of the fundamental principles of biology.

Dysrhaphia is a genotypical manifestation of the individual; *i.e.*, it is a hereditary character inhering in the gene

Man is considered to be one species (Schulte, p 272).

A *variety* is an indefinite, "different" genotypical aspect of individuals of the same species, *i.e.*, a variety is one individual of the species differing genotypically from its kind in one point or in several points.

A *race* is a group of similar varieties; every variety is capable of starting a race in an enclosed area to which he is adapted and given certain groups of external circumstances

Atavism used to be defined as the occurrence of a characteristic observed in more distant ancestors, but not in the more immediate ones. A feature was said to be atavistic if it was exceptional in that particular species, but was typical of another; it was thought to point to descent from the latter. Present-day geneticists do not believe that the occurrence of such a feature – *e g*, hypertrichosis, or a localised white lock of hair – proves that these individuals are descended from a certain animal species in which the feature is common They regard it rather as a chance constellation of the genes which on this occasion happens to be similar to that regularly occurring in other groups Atavism might be briefly described as a polygenic re-combination. or the exclusion of an inhibiting gene

Taking the foregoing into account, we might word Jelgersma's statement differently, as follows: "The dysrhaphic genotype and phenotype within the species of *Homo sapiens* is an individual that, skipping a series, again exhibits a feature which was a characteristic of its distant ancestors, *viz.*, the mammals".

In the modern view, however, if a species exhibits a certain genetic imbalance, this does not imply that that species is descended from a class of animals having the same imbalance. According to Haeckel's fundamental biological law – abandoned by modern biologists –, *viz.*, "Ontogenesis is an abbreviated phylogenesis", Jelgersma's proposition could be formulated as follows: "Dysrhaphic man is an ontogenetic stage of development of the 'normal' phylogenetic type of man", or, "Dysrhaphic man persists ontogenetically at a level of development which, phylogenetically, has on the whole been surpassed", which is only a more involved way of saying that dysrhaphic man's development has been arrested at an early stage. We shall find some support for the hypothesis if we relate the findings in very specific groups in the animal kingdom to man. Man is one species with many varieties but no hybrids in the biological sense of the word; he is one species with a homogeneous "core" by virtue of which he is a human being. This core is cushioned on heterogeneous characteristics.

To our mind, the manifestation of characteristics in a homogeneous species, in which there is only cross-breeding between races and varieties, cannot be usefully compared with the occurrence of identical characteristics in descendants through the cross-breeding of sub-species (mongrels in the animal kingdom). Therefore, the high incidence of dysrhaphia among descendants after the cross-breeding of very different sub-species of the animal kingdom can hardly be adduced as evidence that dysrhaphia in the homogeneous species *Homo Sapiens* is an atavistic phenomenon.

True, some "dysrhaphic" manifestations (non-fused vertebral arches) are common in the early embryonic development of man. If such a condition persists wholly or in part in the adult, this defect is to be regarded as a stagnation in *individual* development. Before we can legitimately conclude that such a stagnation is a phylogenetic earlier stage of our ancestors, we should have to prove that a similar imperfection or defect occurs in a pure species of the animal kingdom as a *characteristic common to that species.* Biologists know of no such species. The hybrids, cross-breeds of sub-species, mongrels, certainly are *not* that species. One might, though doubtfully, consider the progeny of strictly controlled breeding and their descendants to be a pure sub-species created by cross-breeding. Dysrhaphia does occur (more) frequently in it, but not *generally* as a characteristic of that supposed sub-species. We are therefore

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Lorents, Sillevis Smitt have shown that the characteristics of these absolutely or relatively less viable individuals are (dominantly) heritable. Far from dying out, it seems that dysrhaphic man still appears in large numbers after millennia of natural selection. Indeed, it looks very much as if sufferers from dysrhaphia are greatly on the increase, certainly in the outward forms of a heredo-degenerative dysrhaphic constitution, notably the degenerative back in all its aspects.

It is inconceivable that a doomed variety should propagate itself against all expectations, if it is thought to have arisen as an atavistic preliminary stage; once produced and left to itself, the variety must inevitably have become extinct. There is much more to be said for a recurrent genetic disturbance in further development. Degenerative dysrhaphia would thus be a pathological, ontogenetic *over*-development with respect to the average level of development attained phylogenetically. To clarify this we should like to point out that reputable anthropologists (Brown, C. G. Darwin, Eiseley, Cartier) foresee a change in the stature of future man; *e.g.*, they predict that, together with over-development of the central nervous system, there will be regression of the extremities.

Similarly, it is conceivable that, during his development towards his present phylogenetic stature, man, represented by a number of dysrhaphic-degenerative individuals, has already crossed the frontier into a new country. These individuals are "over"-developed in having a hypersensitive central nervous system and a refined build of body, but showing some signs of an ontogenetic preliminary stage persisting in other organs. We say that that build is degenerative because, as we see it today, the defects of the individually persistent ontogenetic preliminary stage make that impression upon us. In actual fact, the individual concerned, as a whole, is supposed to have evolved towards a goal, which is not yet clear to us, where he would no longer need the qualities (firm supporting frame) lost at the time when the defects arose. If we reason along these lines, we may well ask whether it would not be better to speak of "over-cultivation" than of "degeneration".

Is the dysrhaphic constitution by any chance a (first) transitional step towards the future type of man foretold by anthropologists, with even larger cerebral volume, a perfectly functioning central nervous system, with regression of the digestive tract, toes and feet, with weaker extremities and a weak frame? It would be easier to answer this question if it could be shown that the dysrhaphic constitution occurs today as frequently as, or even more frequently than, it did "formerly". Upon enquiry, we were informed by a biologist that it was useless to seek for any proof of general "change" in the human frame from the examination of skeletons taken from tombs a few centuries old. Moreover, it

also debarred from arguing that the (more) frequent occurrence of dysrhaphia in the products of inbreeding in these groups of animals proves that the dysrhaphic animal (man) is a phylogenetic earlier stage, or, to put it differently, that the dysrhaphic is an atavistic variety of the ancestor.

Jelgersma's investigations have established it as a fact that the genotypical disposition to dysrhaphia comes out most clearly under very special conditions of propagation, *viz.*, hybridization and inbreeding. Both in the vernacular and more or less by definition, the words "hybrid" or "mongrel" – in the biological sense of cross-breeding of the species – and certainly "inbreeding", represent concepts which come close to the idea of "degeneration". This is more clearly so with "inbreeding", from which the biologist expects to find *products of degeneration.* For, breeding (selection with inbreeding as the result) points to deliberate refining, completing, perfecting, further developing the species, even if concentrated upon one quality or several. Then, very soon, "degenerative" and "*over*-cultivated" merge into a synonymous concept.

Since, *besides dysrhaphia*, several degenerative features, clearly recognisable as such, occur in animals obtained by the same breeding process – especially in the descendants of pedigree animals and in the Manx cat – it is natural also to regard the dysrhaphic disturbances as the result of a degenerative process.

The foregoing would seem to warrant the assumption that the dysrhaphic in man is degenerative and that the dysrhaphic himself is in a certain stage of his phylogenetic development. The degenerative character of the process offers yet another argument showing that a phylogenetic stage of this kind is an *over*-developed *subsequent* stage, rather than an under-developed atavistic preliminary stage. Thus, for example, dysrhaphia in the Manx cat goes hand in hand with other degenerative features, including the loss of its tail. Where the relationship between the ape and man is concerned, the change from the tailed to the tailless animal is taken to be a development in a positive direction. On this analogy the development of the dysrhaphic tailless creature could be conceived as a degenerative *over*-development.

The dysrhaphic individual is a variety and this variety proves to occur frequently by heredity. In the Darwinian view, the hereditary bearers of this genetic pattern, as an atavistic preliminary stage, would have died out or have been greatly reduced in numbers; for, few dysrhaphics are viable (hydrocephaly, myelomeningocele, etc.), or highly reproductive (meningocele, myelodysplasia, negative natural selection).

There is not a single plausible reason for supposing that the coefficient of fertility of dysrhaphic man is high, high enough to compensate for his diminished viability or procreativeness Investigations carried out by Bremer, Curtius,

arthrotic changes, changes resembling a disc lesion with intervertebral narrowing, or a prolapse of the disc.

We list below the important literature dealing with this subject and it will be seen that opinions differ.

1. "An injury features in the clinical histories of between 50% and 60% of hernia nuclei pulposi patients".
   Armstrong, p. 70, Bradford and Spurling, p. 101, Kostić, p. 74, Krayenbühl, p. 33, Krischek, p. 33, Love and Walsh, see Zaayer, p. 175.
2. "An injury features in the clinical histories of between 20% and 40% of hernia nuclei pulposi patients".
   Ernsting, p. 24, Waris, p. 30, Zaayer, p. 175.
3. Although an injury occurs more frequently in the histories of hernia patients, the following authors found a clear causal relation between injury and hernia in only 10% to 16%:
   Amstrong, p. 70, Cossa, p. 210, Kostić, p. 74, Krayenbühl, p. 33, Roeder, p. 12.
4. The following authors give it as their opinion that "an injury is seldom causally related to herniation":
   Biemond, p. 233, Reischauer, p. 20, Schmorl, p. 161, Zukschwerdt, p. 190.
5. An injury is said by the following authors to lead to herniation only if the disc had previously undergone degenerative changes:
   Biemond, p. 233, Exner, p. 109, Krischek, p. 31, Lindemann and Kuhlendahl, Reischauer, see Zukschwerdt, pp. 190 and 192, Schmorl, p. 161.
6. In the opinion of Bradford and Spurling (p. 117) and Brocher (monograph, p. 37), an injury markedly precipitates back disorders in cases of spines already bearing signs of degeneration or other anomalies.
7. Lewin (p. 52) stands quite alone in his decided opinion that "A trauma is a sine qua non (of low back and sciatic pain). The specific trauma might have been forgotten".

We have stated that the aetiology of low back pain is unknown. By this we meant that the fundamental cause, the link in a chain of causal relations without which back disorders cannot occur, is not known. It is usually very difficult and often impossible to establish a causal relation.

Suppose someone were standing within range of a gun and were hit; what would be the true cause of the accident? Admittedly, the bullet causes the wound, but that person would not have been hit if he had not selected to expose himself to risk, or if circumstances had not led to his being within range of gunfire.

This is clear in the vernacular· If a person takes undue risks and falls a victim to them, it is said to be "his own fault for being so foolhardy" – without reference to the fall itself, or whatever, nor to the injury sustained.

Say that a person out of training lifts a heavy object and strains himself; what then is the cause of the resulting lesion? It is not only the weight, nor the act of raising it. Both the lack of practice and the stupidity of the person

1. There is not always a clear dividing-line between a disc lesion, a hernia nuclei pulposi and spondylarthrosis.
2. If the clinical picture is represented dynamically, it is found that in the long run the disc lesion with herniation is accompanied by manifestations of spondylarthrosis (hooks on vertebral bodies, narrowing of the intervertebral foramina, hooks and bony spurs in the intervertebral canal and in the foramina, posterior lipping inside the vertebral canal). It may be added that, as the clinical picture develops, what was originally an independent spondylarthrosis deformans sometimes exhibits the symptomatology of a degenerated disc, culminating in herniation.
3. In the vast majority of cases, not to say almost always, a disc lesion, whether or not in conjunction with herniation or arthrotic changes, is genotypically determined to a varying extent; which is to say that, from the point of view of the genesis of a disc lesion, the unstable endogenous (genotypical) constitution acts as one of the pathobiological conditions.

One other condition to be noted is a trauma, this being the exogenous factor. Therefore, in every individual case of a disc lesion with herniation and arthrosis deformans, it will be necessary to trace very conscientiously the relationship realised between the endogenous and exogenous factors, by means of a thorough enquiry into heredity and clinical history for details of injuries from earliest childhood right up to the time of the examination.

This is of prime importance where insurance policies are concerned and the grants allowed, since, in the light of the foregoing, it is clearly evident that by no means every disc lesion with herniation qualifies as a case of sickness rather than one of accident. It seems to us that there is always a scale of possibilities, with back disorders contracted upon the least provocation owing to the patient's pronounced degenerative genotypical constitution at one end, and, at the other, those extreme cases in which, thanks to the patient's optimum hereditary disposition, nothing short of a major trauma will produce such disorders.

There is a rich variety of anomalies in the nerve tissue and skeleton of the lumbo-sacral spine. Being part of the constitution, they participate in the interaction between endogenous and exogenous factors and thus play into the hands of the trauma, as it were, in starting off back disorders. Brocher, in his monograph (p. 37) mentions a scale of endogenous disturbances through which the effects of major and minor injuries are heightened. In any event, several anomalies have a stenosing effect; under those conditions, the usual consequences of a trauma, especially those of the chronic minor trauma, are likely to be accelerated, irrespective of whether this happens through spondyl-

through the condition of the subject. This responsibility rests upon society generally, upon the direct employer or upon the insurance company to which the responsibility is transferred by contract. With regard to the conditioned employee or insured person there is the alternative of bearing this responsibility to the full, or of excepting him before the reponsibility is accepted.

These facts suggest several comments.

1. *Can the community legitimately exclude certain of its members from its assumed responsibility for the consequences of a trauma?*

We think not. In our view, any exacerbation of an existing malady brought on by an accident, as well as precipitation of a neurosis in a pre-morbid personality by an accident entitling to compensation, fall within the terms of the responsibility shouldered by society. Not every social system, however, will formulate its obligations in the same way. The aim in the Netherlands is "to insure the worker as he is", Breslau, p. 2336 (Social Accidents Assurance). "Hence the aggravation by a trauma of an illness not caused by an accident can only be accepted if the external event has initiated the sudden development of an existing pathological disposition, otherwise not expected so to develop within the foreseeable future, or has in fact exacerbated it" (Breslau, p. 2338).

In our opinion, this formulation covers disorders of the back brought about by the interplay between constitution and condition and an external factor, like a minor injury, even if the effect of the latter is not decidedly severe or a single one.

Private insurance companies have departed from this point of view, inasmuch as they have adopted what is known as the "normality idea" (sometimes called the "normality fiction"; Schuurmans Stekhoven, p. 875), by which is meant that the person insured against accidents is regarded as "normal" and not ill. An exclusion clause in the policies deprives the conditioned insured person from cover, since it states that the risk does not extend to the consequences of accidents which may also be the result of a disease. We should here make the point that the private insurer, after calculating his premium, is justified in limiting his risks by including clauses in the agreement voluntarily entered into between the insurance company and its client. The difficulty in this matter is not that of establishing the compatibility between risks and premium. The question is, rather, whether it would not be preferable to adjust the premium to the risks, so that it would not be necessary to exclude, say, the exacerbation of a disease by an accident. What is certainly wrong is to couch these clauses in terms incomprehensible to the man in the street. This criticism is apt as concerns back disorders, when they are excluded as "accident resulting from disease" by application of the clause "contracted through an accident in

concerned in performing the action, notwithstanding this lack, are equally responsible for the harm done. The same applies in no less measure to the persistent performance of work that is relatively too strenuous or hard. A sportsman or woman who was able to "go it hard" in early years without undue fatigue and who, for whatever reason, continues his or her pursuits with unabated energy (as a sports teacher or tennis trainer, for instance) in middle age, should not be surprised if he or she begins to suffer from back trouble. This does not only come from the exertion and failing condition, but also, and perhaps primarily, from lack of insight, *i.e.*, failure to realise when the time has come to slip into lower gear.

These examples were intended to illustrate how, in some cases, a given event may be due to a confluence of many causes.

Forensic medicine takes this into account when dealing with problems arising out of accidents. The decisive point in the Netherlands is "reasonable probability", not the "possibility" of a causal relation (Breslau, p. 2336). England and the United States recognise the theory of *causa proxima* side by side with this principle of "the essential contributory cause". German assessors speak of "adequate Verursachung" (Schuurmans Stekhoven, p. 876). These ideas cannot be dissociated from the cumulative effect by which condition plus exogenous factor are together pathogenic, as shown in Speransky's neural theory. They fit in with the ideas on pre-morbid personality and individual pathology held by Siebeck, Adam and Curtius. The connection between these ideas and accidents is explained by Veil and Sturm, pp. 3 and 4.

Our further reasoning proceeds from the following two examples:
A is highly susceptible to a particular innocent substance. B, knowing this, administers it to A for the deliberate purpose of doing him harm. B is therefore guilty. In the victim's condition, the cause of the harm done to him was the otherwise innocent substance.

Some people are hypersensitive to certain medical preparations (Novocaine, serum) which are intrinsically harmless. The medical practitioner, conscious of his responsibility, guards his patient against the possibly lethal consequences of such hypersensitivity by examining him before administering the drug.

In many countries the community has taken upon itself responsibility for the consequences of work done in company employment. These possible consequences may depend both upon the nature of the work and upon the condition of the person engaged in it. His condition may be of a nature to make him vulnerable to work which is innocuous to others. From the moment that responsibility is accepted for the consequences – by social obligation, voluntarily or compulsorily by contract – it also holds for injuries sustained

through the condition of the subject. This responsibility rests upon society generally, upon the direct employer or upon the insurance company to which the responsibility is transferred by contract. With regard to the conditioned employee or insured person there is the alternative of bearing this responsibility to the full, or of excepting him before the reponsibility is accepted.

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conjunction with other *causes*", since back disorders are the product of the interaction between constitution and condition and provocation.

If we reduce this to a formula, we get: Back trouble = constitution + condition + provocation. Condition = constitution + exogenous factors. After substitution the formula becomes: Back trouble = 2 × constitution + exogenous factors + provocation. If a trauma can be proved to be the precipitating factor, the formula becomes: Back trouble = 2 × constitution + exogenous factors + trauma. In this formula the exogenous factors are the unknown quantity, *viz.*, it may have been a disease, but equally well some preceding (slight) (chronic) trauma which brought about the condition.

Often as the constitution plays a part in bringing about disorders of the back, it never causes them. Therefore, the insurer under a policy including the above-mentioned clause stands, according to the letter of the law, to show that the condition of the insured before the trauma was determined by the action of an other than traumatic cause upon the man's constitution. Should it prove impossible to demonstrate such a cause – *e.g.*, a disease – then, to our mind, the above clause does not come into force and the case is reduced to the "result of an accident".

2. *Is it possible to establish, shortly after a trauma has occurred, by what factors the condition was determined beforehand?*

To be brief, we shall sum up previous experience as follows:

a. Demonstrable facts are: residual symptoms of a previous trauma, *e.g.*, impression fracture at the thoraco-lumbar level; functional listhesis, for example, explaining a changed condition at the lumbo-sacral level.

b. The results of chronic minor traumata, *e.g.*, taking the form of spondylarthrotic changes, indented end-plates.

c. The results of infective processes in the spine, *e.g.*, tuberculosis.

d. Rare tumours and diseases of the bone.

e. Manifestations of Bechterew's disease.

The condition is further determined by:

f. Metabolic diseases, including those affecting the skeleton; *e.g.*, osteoporosis.

g. Ageing processes or processes of wear and tear.

h. Degenerative processes.

We have already explained the reasons for assuming that the time when, and the degree in which, these become manifest are for the most part determined endogenously. For instance, if at the age of 40 a person's cartilage has the appearance of that of a 60-year-old, or if a person of 30 has an abundantly developed spondylarthrosis, we say that that person's predisposition to such-and-such was determined endogenously Chronic micro-traumata play the part

of precipitating factors in this process. Attacks of lumbago, crick in the back, low back pain, sciatica, etc. in the past point to the fact that the spine was conditioned by degenerative processes; which means to say: determined by constitution and not contracted through disease. All that an earlier period of back trouble, or even a precedent intervertebral narrowing detected radiologically, shows, is that the patient had had a disc lesion in the past. Not a single plausible reason can be advanced to show why a trauma cannot re-precipitate a "healed" disc lesion or start off a fresh one at another level. In this sense it is difficult to ascertain how the condition arose in a chronic sufferer from back trouble; it is incomprehensible to us that recurrent disorders of the back should so often be regarded as a disease, while the constitutional predisposition to those disorders is so clearly evident.

Lastly, we wish to comment on the fact that clear manifestations of acute and chronic rheumatism are seldom observed in neurological and neurosurgical cases. Rarely indeed could it be shown convincingly to have influenced the condition of the back. In some persons above 50 years of age we found thickly swollen joints at vertebrae L.III – L.IV – L.V, so much so that the spinal canal and the foramina had narrowed as a result. The surrounding bone was then osteoporotic and the joints themselves were either brittle or soft. But we do not see how such a condition can be established without exploration.

Hence, in the event of symptoms of back disorders following a trauma, we do not think it is reasonable to ascribe these symptoms in a person under 50 to an antecedent pathological condition of the vertebral joints, simply because the patient had occasionally had "rheumatic" trouble in the past.

3. *Is it possible to relate the condition of an employee to his or her work?*

Brocher shows that this is possible in a monograph (1957). He predicts that persons who have not had low back pain between their 15th and 25th year are liable to suffer from it if, despite their weak constitution or condition, they are put to heavy work (Scheuerman's disease, p. 22, degenerative changes, p. 24, (postural) anomalies, pp. 23 and 32, residual conditions of infection, p. 43, and of traumata, p. 50). Thus there is a scale of complaints to which a given individual is considered possibly to be predestined. A pre-employment examination (with radiological diagnosis) of the spine reduces the risk of invalidism and lost zest for the employee and the financial risks of the employer, according to Brocher.

On the evidence of an enquiry instituted in American industry, 90% of the industrial accidents in a certain group happens to 15% of the operatives. Apparently there are some people who are accident-prone.

Pre-employment examination has been adopted on a large scale (Allen,

Linden, and others, see Brocher, p. 53). Considerable numbers of those seeking work were rejected (10% to 20%), often on account of degenerative manifestations (disc lesion, transitional vertebrae, etc.). Lewin (p. 91) quotes Stewart as follows: "By pre-employment examinations of stevedores 30 per cent of applicants have been eliminated as unfit for this work; the time lost as a result of backaches has dropped by 82 per cent; and the compensation paid has fallen from about 375 days to 13 days."

These facts do not surprise us; they corroborate our own experience. When persons, discovered in time to be suffering from some sort of back disorder – *e.g.*, the syndrome of the degenerative back – followed our advice as to the kind of life they should live, they were rewarded, even at a later age, by an abatement of their disabilities.

4. *Is it possible, at the time when an injury is suffered, to establish what the antecedent relation was between the condition of the patient and his work?*

Ethical decency towards the employee demands that efforts should be made to discover what kind of work he may reasonably be expected to be capable of performing. If there has been no pre-employment examination and a trauma is suffered, to our mind there is no excuse for deciding, after the event, that the insured employee was, in fact, not insured. Yet this is recurrently happening in daily life, when it is decided, after the event, that before the trauma the patient had a "diseased" back. Indeed, this may be so, though it remains to be seen whether a true *disease* can be proved as cause. Is it not more likely that the preceding state of the back was conditioned on a constitutional degenerative basis? This could not be called a causal disease.

5. *Is it morally defensible to appeal, after the event, to an incongruity between the condition of the employee and the work allotted to him, in order to evade the accepted responsibility for a trauma suffered?*

One's natural inclination is to say that it is not. But the circumstances under which the responsibility was accepted play too important a part to admit of a simple, general answer. When a company compulsorily insures its employee "as he is", this signifies that responsibility is also being accepted for whatever condition the employee may be in; hence also if his back is degenerative to such an extent that no more than physiological strain would be enough to set up disorders. It is not surprising, therefore, that, in countries where a high level of social welfare is organised, there is a perceptible tendency towards erazing the borderlines between the results of accident and of disease, as well as the results of any condition to which the constitution predisposes.

The private insurance company, or the employer, has the right to limit the risks accepted within reasonable bounds, for which purpose he can either

exclude bad risks or raise the premium. We should think the latter is preferable, because it accentuates the social character of the insurance and, furthermore, comes closer to the purpose of the insurance by offering greater security.

Bad risks are excluded by pre-employment examination. The medical examination of applicants for heart and lungs has already been widely adopted. The general tests employed for insurance purposes are quite inadequate to establish the condition of the back; X-ray diagnosis is very seldom resorted to.

It is not a happy solution of the difficulty to refer to the clause stating that, if the condition of the insured person at the time the insurance is entered into is subsequently found not to have been optimal, no benefit shall be due in the event of an accident. First of all, the insured person cannot assess these consequences and, secondly, this clause opens the way to many misunderstandings, since it is doubtful whether a pre-existing condition can later be correctly defined. According to statements made by patients, the least elegant method is regularly adopted in practice, *viz.*, in the absence of a careful preliminary examination of the back, which would have provided the grounds for sound conclusions, the insured person is informed, after receiving an injury, that, on account of the condition of his back, he *did not* qualify for cover against accidents. Or else he is informed that the symptoms produced by the accident resulted from disease, whereas in actual fact the trauma attacked a degenerative back pre-formed by constitution to suffer it in the majority of cases.

6. The refusal to engage an applicant with manifestations of a degenerative back (*e.g.* spondylolisthesis) saves the firm 5000 dollars per person! (Marsh and Rombold, see Brocher, p. 56.)

Let us think for a moment, however, what this means to the victim and then ask ourselves *whether it is morally defensible to force people to undergo an examination, the findings of which may shut them out of the employment they seek.*

Nobody can be in any doubt when it is a matter of life and death to the person concerned (*e.g.*, a disease of the heart) or of spreading infection among others (*e.g.*, diseases of the lung).

At a conservative estimate, the incidence of the degenerative back is 7% to 25% (manifest and subclinical cases respectively). These figures roughly agree with those found in pre-employment examination. We have objections to raise against allowing the consequences of such an examination to come to bear upon so large a proportion of the population, apart from the objections in principle as regards the individual. First of all we wish to point out that we have come to know people with manifest degenerative backs who their lives long had performed excellent work for the benefit of the community under

difficult physical conditions. They would have been prevented from doing this if the advice of pre-employment examiners had been followed. It is suggested that the refusal to engage a person for work beyond his physical capacity may possibly spare him from early invalidism; but the diminished employability to which he is thus doomed from youth upwards should not be forgotten. Naturally, too, this means a financial disadvantage to him, probably more than the 5000 dollars mentioned above during the most productive years of his life. The result of the medical test pursues him from one job to another. As against the safeguard from disabilities and the lack of zest, it has to be remembered that the condemned individual feels the onus of inferiority resting upon him from his youth onwards.

It is a known fact that the very consciousness of having a disorder fosters symptoms and this opens the door to neurotic reactions and other psychogenic disturbances in those predisposed to them. It is, of course, impossible to keep the subject in ignorance of the result of the pre-employment examination, as, even if he is not told by the examiners, his further encounters will confront him with it. An examination of the kind should be extended to many sections of the public and, by rights, should be performed by qualified specialists, which would be unimaginably costly. X-ray examination would be indispensable. We are in no doubt at all that large-scale X-ray screening as a form of pre-employment examination is inadmissible so long as it has not been firmly established that the probability of genetic damage through this exposure can be ruled out.

The character of this examination, attuned as it is to the demands of economy, as well as the social consequences, which interfere with the liberty of the individual and reduce him to a cipher, remind us of Aldous Huxley's "Brave New World". It is as though humanity is helplessly drifting towards a social system which, in some respects, bids fair to make reality of Huxley's nightmare world. It is certainly not the medical practitioner's task to contribute towards its consummation.

With regard to the degenerative back, the above holds in a mitigated form for medical tests on behalf of insurance companies. In the author's personal opinion, the best way to overcome the difficulties enumerated is to raise the premium, *in which case the bearer of a degenerative constitution need no* longer be excluded from insurance against accident risks.

In individual cases, pre-employment advice should, we think, be extended if the patient voluntarily asks for it The admissibility of an individual X-ray examination in support of that advice depends upon the gravity of the clinical history, especially in respect of hereditary taint. In these exceptional cases the

examination will at the same time usually produce evidence for or against medical treatment. The advice itself, which must be tendered with due consideration of the individual's psychology and which need by no means be explicit in regard to his morphological condition, can be compared with the reports issued upon testological psychological examination for choice of vocation.

The problems surrounding trauma as a precipitant of back disorders have existed for longer than a mere decade or so. But familiarity with the clinical picture of hernia nuclei pulposi and the experience gained in the surgical treatment of many thousands of backs have opened up new vistas, through which these problems are now seen in a different light. Current views still derive partly from data recorded at the time when sciatica was still "neuritic sciatica". At as late a date as 1914 Quetsch ("Die Verletzungen der Wirbelsäule durch Unfall" – "Trauma of the Spine") described a narrowed disc as a secondary post-traumatic fused vertebra, whereas" a fracture of the L.V arch" (p. 99) would now be accepted, without much hesitation, as a spina bifida occulta.

It is incomprehensible to us how Vogelenzang, in his "How to Judge whether Certain Disorders are or are not the Result of an Accident", 1950, p. 41, should come to state that "Statistics show that 95% of lumbago cases are infective in nature". The same author writes on page 40 that with a rheumatic lumbago one sometimes comes across several similar attacks in the past, or neuralgic and rheumatic disorders in other parts of the body, in the clinical history (torticollis rheumatica, brachialgia, sciatica, shoulder myalgia, etc.). As though Schmorl had not described the clinical picture of osteochondrosis generalisata several decades earlier! Meanwhile, in a monograph (1954), Exner has thrown much light on the relation between osteochondrosis cervicalis and brachialgia, myalgia and so forth.

It seems as though low back pain is destined to be the subject of much misunderstanding. Baumann's statement in an article (p. 6) on the treatment of sciatica with Tolserone that "there is a growing tendency among surgeons, too, nowadays to shy away from pronouncing in favour of an operation for a possible hernia" is contrary to the actual facts. The Central Statistics Bureau (Utrecht) reports an annual *increase* in the number of operations for hernia during the past few years by 5% to 12% (these figures referring to panel patients only). Armstrong (p. 142) considers infiltrations with local anaesthetics to be a help in the diagnosis of traumatic myofascial strains. "Infiltration of the tender area with local anaesthetic temporarily abolishes all symptoms arising from such an injury". That is true, but paravertebral anaesthetization of a

tender area in the low back likewise abolishes the distress caused by a hernia detected subsequently at operation.

"Bridging symptoms" is the term given to symptoms which persist unchanged from the moment of the trauma and thus bridge the time until it has to be decided whether the condition created is the result of that trauma. A decision of the Central Court of Appeal (1951) has made the qualification of backache as the result of an accident conditional upon the occurrence of symptoms of back disorders (Biemond, p. 234). According to Reischauer, the celebrated German consultant on accident risks, Zukschwerdt (p. 190) and others, a painless interval from hours up to roughly 8 days immediately after the accident cannot be adduced as evidence against a traumatic genesis. In our opinion, the first onset of symptoms after a trauma stands in no significant relation to the genesis, whether called traumatic or not, of a disc lesion; for the protrusion of the disc is a "growing" process; the disc bulges and swells at the site of rupture. Owing to this dynamic development, the clinical picture follows the familiar course, from low back pain to sciatica with or without low back pain. Sometimes the gradual emergence of the disc can be seen during operations and very clearly indeed when, in cases of lateral prolapse *in the lateral recess of the vertebral canal, tissues are removed which had till* then prevented the disc from protruding any further (*e.g.*, fanwise extensions of the pedicles of the arches, especially when the vertebral canal is wide and a flat oval). Moreover, the affected disc may have degenerated pre-morbidly to a varying extent, either parallel with the age of the patient or not, with the result that in one case a prolapse develops suddenly, or early, after a precipitating trauma, while in another it does so gradually, or late.

Finally, we should like to emphasize the following, which represents personal experience gained from the surgical treatment of approximately 2500 patients suffering from one form or another of back disorders.

1. Neither macroscopically nor microscopically does the condition of the extirpated nucleus warrant a definite pronouncement as to the traumatic genesis of the hernia or otherwise

2. Microscopy shows a variety of degenerative changes in the tissue of the disc, which might indicate that the degeneration in this particular disc had been proceeding for an indefinite time

3. Decided signs pointing to the traumatic genesis of a hernia are not found in the macroscopic aspect in the operating field of the laminectomy. A slight tendency to increased engorgement of the vertebral canal might point to it (this in agreement with Schmorl's remarks on the subject, p 197) if the interval between the trauma and the exploration had not been unduly long. As against this, a tendency to engorgement can be explained in various other ways

4. Apart from those rare cases in which the traumatic genesis of lesions of the spine

was established beyond doubt by fractures, we do not know of one case of an uncomplicated hernia nuclei pulposi, in 2000 patients suffering from some form of back disorder, which could *confidently* be proved to have been caused by injury to a previously healthy intervertebral disc.

5. This notwithstanding, the available data have convinced us of the precipitating effect of a trauma upon a disc that has already degenerated.

Summary

A degenerative back gradually sickens under the strain of daily life.

It is very common for a slightly degenerative or already diseased back to succumb to accidental excessive strain, which may, though very exceptionally, likewise attack a perfectly normal back.

A constitutional predisposition can never be the cause of a disease. The pathological condition, therefore, after an injury proved to have been severe enough to produce the effect, should be attributed to an accident in the absence of evidence proving that an antecedent illness was the definite cause of the previously diseased state of the back.

Chapters 6, 7 and 8 in Retrospect

Let us now try to integrate the views expressed in the foregoing three chapters into a single conception.

A typical feature of the endogenously weak back is that its various aspects cannot be expressed numerically; indeed, it is not even an accurately definable clinical picture. This implies that, among other things, the correlation between various aspects and a more general disturbance cannot be traced with precision, because there are multifarious gradations, from "normal" to pathological, of each individual aspect.

In a small number of cases injury is possibly responsible for the pathogenesis of back disorders. Nonetheless, a relationship does exist between the severity of the trauma (exogenous factor) and the patient's condition (ever-changing state of being) at the moment of its occurrence. It is exceedingly difficult to ascertain the degree of degeneracy in a gradation from the optimum to an extremely weak condition of a "normal", or to some extent degenerative, system. The problem becomes more difficult still if the psycho-somatic manifestations of an endogenously weak back suggest a refinement of the human structures, so that the constitutional lability could with equal truth be denoted as "excessively cultivated".

We must once again stress the fact that very many sufferers from spinal disorders are endowed with a constitution which, except for minor subclinical manifestations of the condition described, may fairly be called normal. The

pathogenesis of these spinal complaints is obscure, apart, of course, from purely traumatic cases.

In sufferers from an *obviously* endogenously weak back the signs and symptoms are seen to develop along the lines described below. These dynamisms were already known, at least in part. If, however, a connection can be found between certain observed psychopathological behaviour and abnormal psycho-motoricity, these theoretical formulations gain a firmer basis, at least where the endogenously weak back is concerned.

1. There exists an intimate relationship between the endogenously weak back, with its consequent symptoms, and a certain structure of personality. The latter, too, has an endogenous (constitutional) basis as far as certain constituent factors are concerned.

2. The occurrence both of the disease and the symptoms is associated with the confrontation of specific unsolved emotional conflicts; for there is a close relationship between the psychological valency of the physical apparatus of movement (muscles and joints) and the specific contents of these unsolved conflicts.

3. The disorganisation of the muscle-joint apparatus and any changes in the structure of the apparatus of movement that may ensue are actualised only if the individual having the personality structure referred to under 1. cannot, or cannot adequately, solve his specific emotional conflicts in a truly human fashion. The discharge then shifts from the psychic to the somatic sphere, in the sense of a primitive solution of the conflict, sometimes effected within the endocrine vegetative system.

4. The persistence of unsolved conflicts may evoke unconscious destructive drives which, likewise subconsciously, lead the patient into a state of accident proneness. (The injury implied here may be either the effect of gross external impact or that of strain and purposeless, unco-ordinated movements.)

Certain features are often recognisable as common to patients manifesting a so-called degenerative back: An asthenic habit makes them intensely sensitive to the injury sustained; disorganised psycho-motoricity is more apt to produce unco-ordinated movements placing extra strain on the body, the frequency of which may well be increased by outbursts of aggressive vexation; lastly, patients who lead a psychasthenic or neurotic life generally will suffer more and probably longer from the effects of a trauma.

* * *

All in all, we come to the following *conclusion*: Apart from stigmata degenerationis in the form of psychic disturbances and psychomotor abnormalities – here leaving out of account such degenerative signs as congenital disorganisation of the structure of the spinal column –, regressive tissue decline in the spine is manifested in the following forms:

Degenerative changes in the disc with hyaline degeneration of the tissue and intervertebral narrowing.

Degeneration of the disc in association with hernia nuclei pulposi.

A disc lesion in association with hernia nuclei pulposi and spondylarthrotic reactions of the bone, owing to which the lateral recess, foramina and intervertebral canal in the spine become relatively too narrow.

CHAPTER 9

TREATMENT

General Remarks

As practically no two sufferers from back complaints present the same syndrome, each case has to be judged on its own merits. Sometimes the alleviation of pain is a matter of primary concern, at others the object is to keep the patient going at all costs, even if it means continuing pain or distress. It should not be forgotten that some patients are incurable, at any rate by the means we have at hand. Few patients realise this and it is usually injudicious to put it to them baldly; so, plagued with their disabilities, they go about, despairingly seeking help wherever they think they may find it.

It is not surprising to find diversity of opinion on the therapeutic treatment of back disorders in the literature. It is due to the variety of the manifestations, the different objects for which the therapy is prescribed and the subjective evaluation of the results obtained. Upon the subjective interpretation (both the medical practitioner's and the patient's) of the result depends the indication for further treatment or management. In practice, it is a case of "respice finem".

General remarks on the follow-up

The appeal of a particular form of treatment should be strictly subordinated to the results of a follow-up performed with the utmost impartiality.

The practitioner loses sight of his cured patients, but his stubborn cases continue to preoccupy him; these patients come again and again to his consulting room and induce in him a psychologically understandable affect, owing to their failure to respond to his treatment. A few failures impress him more than any amount of successes.

However carefully his records are kept, details of his patients' condition eventually disappear into the background. We know it for a fact that, at sight of the scar of a laminectomy, the consultant immediately concluded that the patient had been operated upon for a hernia nuclei pulposi, disregarding the degenerative condition or, say, a manifest spondylarthrosis, or even absence of any hint as to its aetiology. It is not only laymen, but quite often some doctors who identify back disorders with hernia, a fact which brings the surgical treatment of hernia nuclei pulposi into discredit.

The good or bad repute in which a given treatment stands comes to the patient's ears, but the uninitiated are not competent to distinguish one case from another; far less are they prepared to accept some causal relation with psychic influences, unsatisfactorily resolved insurance problems and the patient's unconscious wish to be ill.

All these difficulties occur in a mitigated form in organised follow-up. This examination should be performed by someone other than the prescribing therapist, so as to secure it against any bias on his part. Naturally, the objective facts submitted by the consultant have to be incorporated in the records and interpreted in the light of the whole picture. Not infrequently, the reliability of the patient's intimations has to be tested by an examiner trained in psychiatry.

There should be fixed standards for what are called good, moderately good and bad results; the nature of the disorder has to be established; a stated time must be allowed to elapse before a therapeutic effect can be judged.

The imposition of these standards almost precludes comparison between therapeutic results. This applies both to different kinds of treatment and to the same kind of treatment applied by different practitioners under various circumstances.

The results of conservative and surgical treatment are not comparable

This is because the diagnosis of the conservatively treated patient is never certain. From 4% to 10% negative explorations for hernia nuclei pulposi are reported in the literature. If these patients had been treated conservatively instead of surgically, the results of that treatment would have been recorded as related to the supposed hernia. Thus we know of a case treated successfully for years by a physiotherapist; the patient was the show case of physiotherapy for hernia nuclei pulposi. Ultimately, an exploration was carried out when a severe attack supervened, but no trace of a disc lesion could be found.

There should have been a follow-up of about two years before the result of surgical intervention is pronounced to be satisfactory. It is far more difficult to set a corresponding interval for conservative treatment, because this, in fact, never ends. If we ask ourselves (Biemond, p. 243) what is likely to happen to the spine ten to fifteen years after a laminectomy (to which the answer is given in the meantime in actual experience), we should apply the same criterion to the conservatively "healed" disc lesion. It is precisely here that we encounter chronic recurrence and spondylarthrotic changes around the diseased disc.

The results of different methods of surgical treatment can hardly be compared, on account of diversity of surgical technique and of the disorders. It would be futile to arrange the material according to details of technique, because the surgical interventions usually vary in far more than a single detail.

The facts here recorded of our surgical patients were obtained subject to the following criteria:

1. A follow-up period of at least two years after the operation.
2. Neurologists working independently of each other took care of the follow-up anamnesis and the follow-up examination. For the physical examination the examiners had no access to the records of the previous history, the surgical method, the surgeon, nor the follow-up clinical history. A written katamnesic examination provided an insight into the subjective attitude of the patient. To our mind, a patient's favourable reports on his condition are seldom likely to be untrue. Unfavourable written reports, on the other hand, call for a personal check-up of the patient.
3. We adopted the following standards for an evaluation of the results:

Good when the patient, now completely restored to health and without any complaints, returned to his former habits of life.

Good minus when the patient is in health, but has residual symptoms.

Moderately good when, although presenting no symptoms, the patient is *not* in a fit condition to return to his old way of life.

Bad when the pre-operative condition persists, either as to fitness or symptoms.

By whom should back disorders be treated? Or we might put it this way: By whose treatment does the patient and a type of back disorder stand the best chance of being cured? The following chart suggests an answer.

"Diseased Back"	*Symptoms*	*Treatment*
		General practitioner
Constitution	Metabolic	Specialist internal diseases
+		Rheumatologist
Endogenously		
determined		Orthopaedic surgeon
condition	Morphological	Neurosurgeon
+		
Precipitant	Functional	Neurologist
	Psychic	Psychiatrist

The general practitioner will be able to cope satisfactorily with the majority of back disorders, but, if the symptoms persist, the question arises, to what specialist the patient should be referred (Nederlandsche Tijdschrift voor Geneeskunde, 99 (1955) 2669). A tentative anamnesis and examination should supply the answer. If the patient presents symptoms of general ill-health, or if

there are reasons to suppose that he or she is suffering from an internal complaint, the obvious course is to call in a specialist in internal diseases, general surgeon or gynaecologist. If there are indications of rheumatic trouble, the patient complaining of his back would be well-advised to consult a rheumatologist. The vast majority of sufferers from back disorders either complain of low back pain, in which case they should be sent to an orthopaedic surgeon, or else they present a complicated symptomatology including psychic disturbances, radiating pain to the legs or neural symptoms, in which event expert examination by a neuro-psychiatrist is required.

To our mind, the neuro-psychiatrist is the only specialist qualified to deal with the differential diagnosis of a complicated neuro-psychic disorder. This view rests on the experience of numerous cases, the symptomatology and morphology of which form the subject of this study. It is no less reprehensible for an orthopaedic surgeon to operate for a neuro-psychic syndrome than for a neurosurgeon to perform a fusion operation as a "remedy" for low back pain.

Laminectomy requires technical skill (Biemond, p. 243). The skilled surgeon who performs laminectomies regularly acquires experience and practice. He may be confronted with the unforeseen during operations; quite often the pre-operative plan has to be changed; a simple herniatomy may well be complicated by a rhizotomy, or an intradural exploration may be indicated. The specialist discipline, training and practising for the treatment of the nervous system has, since the discovery of hernia nuclei pulposi as a clinical picture, included this in its curriculum. Through this, the practised neurosurgeon has the required experience in the diagnosis and in the surgical technique for the treatment of back disorders caused by a lesion in the spinal canal.

The following considerations may help in the choice of a specialist for the treatment of a diseased back complicated by neural disturbances. Both the general practitioner and the layman know or feel intuitively that there is a temperamental difference between the naturally conservative neurologist and the naturally active surgeon. The inclination will be to choose the more conservative-minded of the two if advice for conservative treatment is expected, or if the practitioner himself considers it advisable. But, if conservative treatment has failed, or the severity of acute neural loss symptoms seems to press for urgent surgical intervention, it is obviously the neurosurgeon who should be consulted.

Conservative Treatment

The problems surrounding conservative treatment could be discussed at great

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Good minus when the patient is in health, but has residual symptoms.

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Endogenously determined condition	Morphological	Orthopaedic surgeon Neurosurgeon
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Precipitant	Functional	Neurologist
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Joint (if blocked) released.

Dehydration of tissues.

Change from a fresh disc lesion to the more physiological form of disc degeneration.

Reduced protrusion of the prolapse.

Attempts by reactive bone proliferation to strengthen the spine locally.

Relapses after conservative treatment are mentioned frequently:

Bradford and Spurling	40%
Ernsting (p. 34)	80%
Love and Walsh	48%
Junkersdorf	15% (within 1 year)

It is seriously doubted whether a disc lesion ever is cured by conservative treatment (see Armstrong, p. 73, Lewin, p. 735, Reischauer, see Schmorl, p. 165, Thurel, p. 25, Zukschwerdt, p. 80). The disc becomes a dead organ, the intervertebral space narrows, the joints undergo changes, the foramina become narrower, the margins of the vertebrae form spondylarthrotic hooks.

When exploring for the cause of acute exacerbation, after a history of chronic symptoms, it is by no means rare to come upon a fresh disc lesion accounting for the acute syndrome at one level, while the remains of a so-called "healed" disc lesion are found at another level. The lipping around an earlier disc lesion thus consolidated is a spot of least resistance. There is now some relative stenosis, which need be little more in some cases to cause root compression. Under favourable circumstances, the "healed" disc lesion may consolidate without leaving residual symptoms, one of those favourable circumstances being a large enough spinal canal to relieve the effects of the stenosis referred to. If, through ossification, the healed lesion has formed a synostosis, this need not cause low back pain, but if the spondylotic changes cause chronic movement of the spine in an unphysiological manner, local strain will result, entailing low back pain and spondylarthrosis which is no longer limited to the affected place. The physician has to look to the future well-being of his patient and, where that is concerned, temporary pain is, to our mind, a secondary matter. This involves keeping a watch on the function of the spine after conservative treatment and, if there is any abnormality of posture or movement, this should be corrected by physiotherapy or, at the worst, by surgical intervention.

There are many ways of applying conservative treatment. Krayenbühl writes (p. 55): "There are many forms of conservative treatment and they are constantly changing. Methods which were much in favour years ago have since been abandoned and new kinds of treatment are being adopted". This plainly

length, but we have no intention of doing so. After a few preliminary remarks, we shall consider the results of some of the more usual methods of conservative treatment.

Conservative treatment is management without an established diagnosis. This deficiency is sometimes mentioned in statistics (Posthumus Meijes), but in others the fact is overlooked that quite a considerable percentage of cases stated to be "conservatively cured hernia" were actually lumbago, a weak back or a spondylosis deformans (Kirschek, p. 65, Spiegel, 1955, p. 348). The purpose of conservative treatment is to vanquish the symptoms of back disorders empirically, irrespective of whether they are produced by degeneration of a disc or by a combination of several abnormalities. In this it succeeds to an appreciable extent, as the following percentages taken from the literature show:

Friedenberg (see Spiegel, 1955, p. 348)	47%
Hamby (see Spiegel, 1955, p. 348)	30%
Junkersdorf (p. 266)	51%
Krischek (p. 65)	74%
Posthumus Meijes (p. 407)	66%

These results were obtained by conservative treatment for a period ranging from *weeks to months*, during which the patient was *not* as a rule *fit to do any work*; moreover, at the end of this period the majority of patients were advised to go slow, which meant that they were unable to return to their normal way of life.

Some practitioners (Junkersdorf) advise conservative courses of treatment so specialised as only to be practicable in a clinic; they are, moreover, very costly This means that a great many sufferers from low back pain have to forego the benefits of such treatment.

Sometimes conservative treatment becomes the turning-point in a patient's career because, as far as his mode of life is concerned, he is compelled henceforth to go at a slower pace. Armstrong (pp. 163, 167 and 169) enumerates the restrictions to be imposed upon the activity of conservatively treated patients for months. Lewin writes ironically (p. 812): "*Conservative* treatment, that seriously alters one's life, is very *radical* in so far as the patient is concerned"

The following factors are responsible for the encouraging results of conservative treatment·

The disappearance of the "oedematous" "reactive" inflammation of nerve tissue, ligaments and membranes.

Break in the vicious circle of pain and muscle spasm.

Resumption of the recovered muscles' task as components of the skeleton.

certain diagnostics. Before explaining ourselves further, let us record the following investigation, on the basis of which this view was formed:

From among several thousand cases of hernia verified at operation, 300 were picked out at random to test the influence of six to eight weeks' bed rest. In 90% of the cases the rest-cure was found to have been in vain. It cannot be ascertained from these records how many *had* been permanently cured by this rest, but what the evidence does show is that there was a very large number of cases in which the surgically confirmed hernia had not responded to rest.

With the object of probing further into this matter, we made systematic enquiries at the polyclinic to find out how the patient had been treated in the first instance. 50% of 200 patients suspected of hernia nuclei pulposi on polyclinical grounds had already had a rest-cure for at least six weeks. The very fact that they came back for treatment proves that the rest-cure had been in vain. We likewise enquired into the fate of patients whom we ourselves had advised to undergo a six weeks' rest-cure, probably on account of a hernia nuclei pulposi. We found that 24 of 50 patients – that is just less than half – had not benefited permanently from it.

Disregarding the statistical imperfections of these enquiries, the figures show that bed rest was effectual for about half the patients suspected of hernia nuclei pulposi.

Two hundred patients were asked how the prescribed directions for the rest-cure had been carried out. It was far easier to get the truth out of those of the patients questioned by someone other than the doctor who had prescribed the rest-cure, when it transpired that 60% had not kept to the "rules". Most of these people had merely lain on a hard mattress for two weeks, without getting out of bed at all, after which, against the advice of the doctor, they had become mobile prematurely; without the doctor's knowledge they had walked about the room or had been compelled for household reasons to leave their bed. The patients were obviously ashamed to admit that they had not obeyed orders, not daring to confess as much to their own medical adviser.

When we enquired into the results obtained in the remaining 40% who had followed out the instructions to the full, we found to our astonishment that they were much the same in both groups About half of each group had benefited by the treatment, either *carried out faithfully for the full six weeks, or less commendably for two weeks.* These facts corroborated the opinion we had formed of the rest-cure.

Excepting cases of spondylarthrosis and low back pain with a marked postural anomaly when the patient is supine, we expect satisfactory diagnostic and therapeutic results from absolute bed rest for *two weeks*! If, after this period of enforced rest, the patient's symptoms have clearly abated, the indicated procedure is to consolidate the improvement by a continuation of the rest-cure in a mitigated form for two to three weeks. Should the same radicular pain continue to radiate and the back still be locked in a marked degree upon trial mobilisation after the first two weeks, there is, to our mind, so little likelihood of a permanent cure that it would be merely loss of time to continue the treatment any longer. This applies to the patient's management at home.

underlines the fact that all the methods hitherto employed have proved to be unsatisfactory in some way or other.

I. Rest

The simplest form of treatment is rest. Presumably, there is no kind of back complaint that does not respond favourably to it, except pain caused by a neoplasm. The symptoms of manifest hernia nuclei pulposi abate the moment the patient is able to lie in bed without muscle spasm. In approximately 7% of the cases subsequently presented for operation it was found that rest did not diminish the pain at all, sometimes, indeed, exacerbating it.

Rest for low back pain has to be prescribed with some circumspection. There are some callings so beset with stress and turmoil as well as social demands, that a *few days of prescribed rest* is the sufferer's one and only opportunity of getting off the treadmill. Happily, nature is able to heal many ills by rest, without needing an exact diagnosis of the ill. To our mind there are a great many people plodding on who could well do with a few days' rest now and again, apart from their annual holidays. If, however, the symptoms are severe, or it is a matter of yet another relapse, it is necessary from the outset to differentiate according to the age of the patient. Remembering the adage that "to rest is to rust", the wisdom of prescribing a long rest-cure for a patient suffering from *spondylarthrosis* is questionable. Rest likewise relieves such a patient's distress, but, after an initial improvement, the symptoms are known to return in a heightened degree when the patient is again mobilised. The mistake can be avoided by taking the patient's age into account and correctly interpreting the syndrome, which is fairly typical for spondylarthrosis.

It is also a professional error to prescribe prolonged rest for those patients who, when supine, are unable to relax their muscles. Persistence in this cramped attitude may well set up chronic malformation of the soft bone of the vertebra. If the patient becomes more relaxed after some time, the harm may already have been done, even though the treatment *appears* to have been successful because the initial pain has ceased.

Opinions differ as to the proper treatment for *acute low back pain*. The predominant tendency is to try out conservative treatment before resorting to surgery. The exceptions can best be dealt with when we come to discuss the indications for surgical treatment, but we feel we should now bring forward our reasons, gathered from our own experience, for calling into question the advisability of prescribing the *classical six weeks' bed rest* as a routine procedure. By it, a sound therapeutic measure is degraded to un-

certain diagnostics. Before explaining ourselves further, let us record the following investigation, on the basis of which this view was formed:

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Hospital nursing may produce more encouraging results, but the number of patients so treated is too small to provide evidence one way or another.

Hence it can be safely said that low back pain is curable by the classical form of bed rest properly carried out, even if the patient presents the symptoms of a hernia nuclei pulposi; but it is impossible ever to foretell what the result will be. If the patient's psyche lends itself to the treatment, *i.e.*, if he has a phlegmatic temperament; if home conditions are favourable; if he can spare the time; if he has sufficient means; if it is certain that he will not have to do any really hard work for two years after the cure... then he stands all the better chance of being cured. Before prescribing a rest-cure on these lines, it is well to consider whether these conditions obtain.

The result of strictly enforced bed rest for two weeks provides the physician with reliable data for diagnosis and for advice as to other kinds of treatment. It also obviates the unnecessary withdrawal from the country's economy, for weeks on end, of a large percentage of the labour force with low back pain, for no well-founded reason and to the patients' own physical and psychical detriment.

II. Radiation, medication and infiltrations

We tried to discover whether backache responds to diathermy and medication, combined or not with bed rest. We cannot give an account of all the methods and shall mention only those of which particulars are known to us sufficiently from the literature and from practical experience. The uncertain factor in all these kinds of treatment is the extent to which the suggestive element reinforces their effects. That it does exist has been proved by experiments. Armstrong (p. 71), referring to it, says that if rest is combined with some form of physiotherapy, any subsequent remission of symptoms is apt to be attributed, probably without justification, to this more spectacular part of the treatment.

X-ray treatment is sometimes prescribed for chronic low backache due to spondylarthrosis. Its results are negligible. It has been stated elsewhere in this book that, in an experiment with two groups of patients, for one of which the X-ray apparatus was switched on and for the other was not, the results of the irradiation and mock-irradiation were exactly the same.

It would seem that, except for unambiguous muscular pain, *diathermy* has little more than a suggestive effect.

Ultrason. Apparently, little success has attended this treatment in practice. It would seem that the suggestive effect of the treatment is in inverse proportion to the patient's familiarity with its name.

Medication. In the acute phase of the locked back the main object is to relieve

pain and muscle spasm. What sedatives or analgesics (morphia, tolserone) are used for the short period in which they are needed is a matter of minor importance. In the chronic stage the object is, rather, to abolish any "reactive inflammatory factor" there may be (salicylic preparations, etc.). Clinical enquiries were made to discover whether dehydration effectually diminishes the assumed oedema of nerve tissue, joints, ligaments and membranes (euphylline). It transpired that this expedient produced no better result in patients at rest than in the controls. Preparations like irgapyrin and butazolidin were used, with varying success, to combat pain in the joints. In some cases, local treatment with cortico-steroid seemed to produce promising results; but it is difficult to reach the spaces between the vertebral joints. Epidural administration has the advantage of reaching the roots as well. If not contraindicated by pre-existing bacterial processes, for instance, this method bids fair to provide a means of influencing non-mechanical root irritation. Although the intrathecal administration of corticosteroid has meanwhile been reported as beneficial for inflammation of the nerve tissue (Lohman, p. 2836), it should only be resorted to with the utmost circumspection.

At the Third European Congress for Rheumatism (1955), Bywaters, referring to a critically selected material, spoke about a comparative clinical examination of acute and chronic polyarthritis. Neither after six months nor after twelve was there any convincing difference to be observed between the patients treated with hormones (Acth and cortisone) and those treated with acetyl salicylic acid. The permanent results obtained with either were virtually of the same value.

The infiltration of Procaine is resorted to with varying success for the alleviation of local painful muscular spasm or tender areas. According to Armstrong, the cessation of symptoms would point strongly to the traumatic origin of the lesion. Infiltrations around and in the vertebral joints have proved to be of some use. We shall deal presently with the application of Procaine to scars or elsewhere subcutaneously.

Procaine blockage of the lower lumbar sympathetic trunk scarcely qualifies for the alleviation of backache; it calls for a special indication.

Opinions differ as to the effectiveness of *epidural Procaine infiltrations* (or presacral injections). Armstrong writes (p. 174): "It is very doubtful if such an injection has any effect at all". Thurel (p. 24) believes that it abolishes the oedema.

We were particularly interested in this matter. It has been our practice to inject 20 c.c. of 1% Procaine epidurally once or twice a week, we considered it best to inject the fluid under pressure to help it to spread around the root in the lateral recess, besides which, the patient then experiences certain sensations (sometimes painful ones) and

these are favourably suggestive. It is sometimes claimed that the fluid thus introduced breaks down adhesions, but, in view of their toughness and tenacity as found at operation, we are somewhat sceptical about this.

One injection is not expected to do much; we do not look for really promising signs until three weeks after the first injection or a few weeks after the termination of a six weeks' course of injections. We verified results six months or longer after an epidural injection course, for which we divided the patients into the following groups:

a. Patients with indeterminate backache and vaguely defined radiating pain in the legs, not suspected – by polyclinical standards – of suffering from hernia nuclei pulposi (60 cases).

Age	*Number*	*Reasonably good*	*Moderately good*	*Bad*
15–30	17	12	3	2
30–40	27	15	4	8
40–50	12	5	4	3
Above 50	4	4	–	–
		60%	18%	22%

Hence the result was not unsatisfactory.

b. Patients with decided radicular syndromes (47 cases).

Of these, 2 patients finished in very good condition,
8 patients showed reasonable improvement;
37 patients showed little or no improvement.

c. We compared the effect of epidural injections upon a pre-operative hernia syndrome (24 cases) with that upon post-operative recurrent radicular symptoms (21 cases):

	Patients not operated upon	*Patients operated upon*
Good results	–	2
Reasonable improvement	5	3
Little or no improvement	19	16

Hence there was no evident correlation between the patients' having or not having been surgically treated and their improvement through epidural injections. The above figures seem to show that such injections do not affect a hernia syndrome in any way worth mentioning

d. Another point enquired into was whether the condition of the surgically treated patients affected the result of the course of injections in any way. For this purpose we divided these patients into three groups corresponding to the gravity of the symptoms persisting after the operation

Result of operation	*Post-operative result of course of injections*	
Good; vague complaints 9	Good	2
	Moderately good	2
	Bad	5

Moderately good	12	Good	1
		Moderately good	1
		Bad	10
Bad	None treated	——	

e. There also proved to be no clear correlation between the gravity of the clinical picture and the result of the injections, as may be seen from the following (46 cases):

	Result of course of injections	
A. Unconvincing radicular syndrome	Good	2
	Moderately good	3
	Bad	15
B. Well-developed picture	Good	–
	Moderately good	5
	Bad	18
C. Complete picture	Good	–
	Moderately good	–
	Bad	3

f. Seeing that a course of epidural injections is specially recommended for arachnoiditis, we examined 6 patients who had been diagnosed as having lipiodol arachnitis and had undergone this treatment. We found no outstanding improvement, it being reasonable in the case of two patients and little or none in four.
g. We had radiographs of 31 of the patients who had had a course of epidural injections. Nine of these patients presented the signs of a degenerative back taking the form of various congenital anomalies of the lumbo-sacral spine. The results in these nine patients were as follows:

1 good
3 reasonable improvement
5 little or no improvement.

The value of this investigation is circumscribed by the small number of patients examined It is perhaps permissible to deduce from it that no clear correlation was to be seen between the findings and the effect of the treatment. We suggest that the following may be inferred:

Low back pain that has no clear-cut mechanical aetiology is reasonably responsive to the treatment

In the presence of a radicular syndrome pointing significantly to a hernia nuclei pulposi, the effect of epidural injections is negligible.

If symptoms persist post-operatively, the treatment has little effect.

Our personal opinion of a course of epidural injections may be stated as follows:

It is well worth while trying for patients with vaguely defined low back pain and equally vague radicular pain, who have not been surgically treated

these are favourably suggestive. It is sometimes claimed that the fluid thus introduced breaks down adhesions, but, in view of their toughness and tenacity as found at operation, we are somewhat sceptical about this.

One injection is not expected to do much; we do not look for really promising signs until three weeks after the first injection or a few weeks after the termination of a six weeks' course of injections. We verified results six months or longer after an epidural injection course, for which we divided the patients into the following groups:

a. Patients with indeterminate backache and vaguely defined radiating pain in the legs, not suspected – by polyclinical standards – of suffering from hernia nuclei pulposi (60 cases).

Age	*Number*	*Reasonably good*	*Moderately good*	*Bad*
15–30	17	12	3	2
30–40	27	15	4	8
40–50	12	5	4	3
Above 50	4	4	–	–
		60%	18%	22%

Hence the result was not unsatisfactory.

b. Patients with decided radicular syndromes (47 cases)

Of these, 2 patients finished in very good condition;
8 patients showed reasonable improvement;
37 patients showed little or no improvement.

c. We compared the effect of epidural injections upon a pre-operative hernia syndrome (24 cases) with that upon post-operative recurrent radicular symptoms (21 cases):

	Patients not operated upon	*Patients operated upon*
Good results	–	2
Reasonable improvement	5	3
Little or no improvement	19	16

Hence there was no evident correlation between the patients' having or not having been surgically treated and their improvement through epidural injections. The above figures seem to show that such injections do not affect a hernia syndrome in any way worth mentioning

d. Another point enquired into was whether the condition of the surgically treated patients affected the result of the course of injections in any way. For this purpose we divided these patients into three groups corresponding to the gravity of the symptoms persisting after the operation

Result of operation		*Post-operative result of course of injections*	
Good, vague complaints	9	Good	2
		Moderately good	2
		Bad	5

either on account of their age or their complaints, we recommend starting the treatment with a course of vitamin D repeated every six months.

It is impossible to establish the nature of the numerous hormonal deficiencies, but involutional osteoporosis often manifests itself in women during or immediately after the climacteric. The indicated course would therefore seem to be to rectify the assumed hormonal discrepancy by substitution for the lost function of the gonads. No sound reason can be advanced, however, against the possibility that the whole involutional process is being induced at a higher (endocrine) level integrally, eliciting other involutional conditions (osteoporosis, greying, ageing of the skin, etc.) together with diminishing function of the gonads. Some advise making up for the reduced hormonal function of the female gonads with an excess quantity of male hormones, to which are added small amounts of female hormones. This has already been fully discussed in the chapter on involutional osteoporosis. We applied this treatment to 19 patients (between 40 and 52 years of age) for a whole year, without achieving in a single case the startling success claimed for it. The continuous nagging low backache and the tenderness of the superficial parts of the skeleton to pressure persisted on and off; nor did they disappear when a shot of Vitamin D was added to large doses of neosterone or durabolin. What we did find was that it did seem to give the patients some sense of well-being. Eight patients said that they felt less "insupportably tired". The menstrual cycle was disorganised three times, transiently accompanied by fairly severe abdominal pain.

Even if no cure was brought about in these cases, one wonders what the patients' condition would have been without treatment. For, involutional osteoporosis can lead to strongly progressive symptoms, ultimately culminating in serious deformity. These 19 patients were already in such an advanced stage that, on account of radicular symptoms, they were referred to the neurosurgeon for his opinion as to the possibility of surgical therapy. Far better results are to be expected if the disease is diagnosed in good time and the diminishing hormonal functions are bolstered up prophylactically, as it were. This entails recognition of the type of constitution of the prematurely ageing human being; and in this, family clinical records can be of the utmost assistance.

This affliction deserves greater attention, not only because of its incidence, but also because of the untreatability of the manifest condition and the disabilities which lead to a high degree of social invalidism. The serious consequences of letting the chance go by of applying prophylactic treatment are sufficient in themselves to justify its application in doubtful cases. For these reasons it has become our practice to prescribe unremitting medication with

The pre-operative course of injections is a rewarding, suggestive treatment *ut aliquid* for the period during which the patient is under polyclinical observation. There may be a spontaneous cure during this period and meanwhile the patient is being watched for hints of the development that will suggest what further therapeutic measures are to be taken. The advantage of this procedure is that, in the interim, the patient can be encouraged to perform certain physical exercises and to go on with his work.

What has been said in the preceding paragraph applies equally to post-operative signs and symptoms.

We only saw promising signs of therapeutic effect when the injections were given to an operated patient who, after a period free from symptoms, had suddenly been assailed with radicular pain following influenza, a cold or serious chill. We believe such recurrent symptoms are caused by a reactive inflammation, which is apparently responsive to the epidural injections. We had not taken this into account when grouping our material; besides, an enquiry of that kind would only have had any purpose if there had been a control group of patients who were *not* given injections under these circumstances.

Some forms of low back pain are induced by diseases of the internal organs, such as deranged metabolism, gynaecological disorders, gall-bladder, Basedew's disease and so forth. Obviously, the treatment of those diseases is the primary matter, but they may leave residual conditions which could be regarded as *deficiency diseases* in the broadest sense (calcium, proteins, vitamins, hormones, etc.). As far as is known, the effect of these deficiencies on the spine is to soften the bone and widen the mesh of its structure. At certain periods of life this is called premature involution, even if the osteoporosis usually associated with that condition is by no means demonstrable. The pain from pressure upon the superficial parts of the skeleton is a clearly indicative symptom, not to say characteristic, of the condition.

It is of little avail to make up for calcium deficiency merely by administering large quantities of re-absorbable calcium unless other deficiencies in the deranged calcium metabolism are made good simultaneously, as was fully explained in an earlier chapter. An appropriate diet will supply a sufficient amount of re-absorbable calcium (milk products, especially buttermilk). In the event of undernourishment, or other abnormal conditions, the diet will have to be supplemented with proteins. Avitaminoses are dealt with in the usual way; the absorption of Vitamin C is assured, in the chronic phase at least, by a proper regimen. As, however, sufferers from involutional osteoporosis all too often neglect to take full advantage of the healing power of the sun,

3. Some patients were advised to wear it periodically, for instance if they knew in advance that work they would have to do was going to put a strain on the back.

It is unnecessary to add that it need not be worn while doing prescribed physical exercises. We advised wearing it during the post-operative period under all conditions for which the stability of the spine was considered to be either chronically or transiently inadequate. Such cases were asthenics, *i.e.*, patients whose muscles were underdeveloped before the operation, people with very long backs (genuine lumbarisations) and several cases in which a hernia nuclei pulposi at the level of the L.II – L.III or L.III – L.IV disc had been removed.

The advantages of this lumbar corset are obvious: it is light to wear and inexpensive; the patient can therefore have several, which is far more hygienic. It is, moreover, inconspicuous. Apart from the actual reinforcement of the skeleton which the corset affords, it gives the patient a feeling of support, comparable to that of placing a hand at his back. It does not hold the wearer stiffly, but leaves a certain amount of freedom of movement; and it does not disable the patient from work. Its purpose is to restrain abnormal movements, so it need not constrict; though not rigid, it signals a warning to the wearer who is about to make exaggerated movements.

Once he has become accustomed to it, the patient not yet surgically treated is often loath to part with it. There is nothing against his wearing it always, because it does not take over the function of the muscles; it only directs them. About six months after the operation, we advise the patient to discard the belt, presuming that, after this lapse of time, a normal muscular system will be capable of supporting the spine sufficiently.

IV. Physiotherapy

We have just stated our case against prescribing a rest-cure unless this is clearly indicated. Its social and personal drawbacks are obvious. It is all the easier to adhere to this principle if, in practice, there is an alternative to hand which is, to say the least, equally effective. In the less serious cases, we urge our patients after a couple of weeks' rest to return to their normal way of life by degrees. A rigid rule of bed rest of two (to three) weeks is prescribed for patients with severe low back pain or sciatic pain, but after this period mobilisation is tried out. If the symptoms persist, little is to be gained, in our opinion, by keeping the patient in bed any longer, but if they abate or disappear altogether, there is some useful purpose to be served by prolonging the period of rest by one or two weeks, with the intention, however, of beginning to

aneurin, nicotinic acid, vitamin D and ovarian preparations. After treatment for the acute exacerbation of back disorders in whatever form, this medication is prescribed for persons presenting signs of premature ageing. What remains to be seen is whether the success of this precautionary measure will ever be definitely demonstrable.

III. Immobilisation of the spine by a jacket

There are, indisputably, a great many patients who experience so much support from the wearing of a corset that they are no longer able to do without it. But the doctor has to decide when it should be prescribed and when not.

In our own practice we have never immobilised the spine by means of a *plaster jacket* which the patient can wear for months on end, either before or after operation. Yet, as will be shown later, our results do not compare unfavourably with those generally reported, more especially in series in which plaster corsets were prescribed post-operatively. Surely it is inconsistent to continue to prescribe plaster jackets if the results are not strikingly superior to those obtained by treatment without such jackets? We do not wish even to try out this method; both mechanically and functionally, immobilisation of that kind must restrict the physiological scope of movement of the spine.

The big orthopaedic jacket

1. In certain respects, what has just been said holds here.
2. The patient calls this contrivance "harness".
3. The patient wears it unwillingly, if, indeed, he does not throw if off within a few weeks.

More patients than we think only wear this "harness" when they have to go for a check-up to the doctor who prescribed it, as we have been able to verify. Admittedly, there are some who derive benefit from it and would therefore be reluctant to part with it; it is up to the patient to weigh the advantages against the disadvantages. But we wonder whether they could not be helped in some other way.

The small orthopaedic corset (canvas belt) with supporting pad for the back

We have prescribed this simple belt pre-operatively quite often under the following circumstances.

1. To relieve low back pain if the stability of the spine left something to be desired.
2. In cases stubbornly resistant to treatment when surgical intervention was contraindicated.

of course; the patient should go to work tentatively every time, just beyond the limit at which movement becomes painful or irksome. The exercises entail no expense and can be carried out by the patient himself and there is no reason why he should not make them a daily habit always; they are preventive against recurrency. Moreover, these exercises, performed upon medical advice, have a stimulating effect; they increase his self-confidence and he feels that he has regained his health. Lastly, through these therapeutic exercises the patient approaches those physiological conditions under which he is presently to resume his former occupation and habits. Hence, if they result in the disappearance of the symptoms, the patient is at the same time re-adjusted and there is no necessity for a further transitional period between the inactivity imposed by his illness and the resumption of work.

The patient can be assisted by a qualified physiotherapist, who knows what to look for and can correct a disorganised body-plan. Depending on the patient's psyche, a more individually directed Mensendieck method may be preferred, especially if a female patient feels attracted to rhythmic forms of physical culture or if the patient has an aversion to simple gymnastic exercises.

The effect of *massage* is overrated. If low back pain yields to gentle massage of the muscles, there is good reason to assume that there is no lesion of the spine. When medical gymnastics or physiotherapy in some other form is recommended, it is as well to point out that this does not include massage. This is undertaken, without a doctor's advice and without success, often for several months, probably because the patient likes it. Massage is only indicated if atrophy or paresis stands in the way of active exercising, but this seldom occurs in unoperated cases, because pareses and atrophies are indications for surgical intervention.

V. *Some forms of special treatment*

1. It appears that the *removal of tonsils and dental treatment* are losing favour as a means of combating chronic arthropathy (Zukschwerdt, p. 153).

2. *The cellular therapy as is advised by Niehans* advocates the administration of cell suspensions from hypophysis, suprarenal gland, liver, etc. It is alleged to cure, not only acute infective disorders of the joints, but also chronic arthrosis, metabolic anomalies and chronic degenerative processes. In view of the successes claimed for it in the propagandistic literature, it is strange that one should never come across a patient in one's practice who could show that he or she had been cured by it. And this is not just our own experience, it is the general opinion expressed by colleagues. All in all, the ratio of this treatment is by no means established.

mobilise and exercise the patient without further delay. In following this plan, the doctor should be fully aware that the therapeutic possibilities have to be assessed for each individual patient. Some patients have the right temperament to submit to a strict regimen conscientiously, whereas other patients have it in them to recover entirely *without rest* when the acute exacerbations have passed.

Contacts with foreign patients and medical practitioners brought us to realise that, to some extent, these traits of character are national; there can be little doubt but that the laws prevailing in a country and its code of behaviour either strengthen or weaken these traits of character. What we mean to say is that it will sometimes be easy to persuade a patient of a certain upbringing to resume work or do exercises in spite of the pain, while another will set his face against all efforts to bring him to lead a more active life until he himself wishes to, usually because he becomes bored.

From this point of view it would seem unwise to enforce the rules of a certain kind of therapy, which have been developed within the framework of the social system of one country, to the nationals of another country without regard to their psychology. An advice to return to work stated as "you must" is itself informed with a therapeutic effect in a German's ears; a Dutch workman would resent it, or would retort that he would prefer trying a few weeks' rest.

Our reason for prescribing exercises after a brief introductory period of rest is that just as many patients seem to recover in this way after the cessation of acute symptoms as after a prolonged, strict rest-cure *not* followed by exercises.

The exercises we prescribe consist of certain movements designed to make the back flexible. (The therapeutic aim is predominantly to combat stiffening of arthrotic joints; as from middle age, we do not think there is any spinal disorder which does not involve some degree of change in some or other joint.)

The performance of movements gets the muscles into good condition again and they are able to participate in the function of the supporting frame. Asthenics, in particular, benefit by this treatment.

The preceding disorders upset the body-plan; postural changes remain, seen in a so-called projecting hip or rigidity of the back. These things bring about cumbersome mechanisms, which should be got rid of at the earliest possible moment to avoid worse detriment to the bone of the spine.

In view of the diversity of factors inducing anomalies of posture and movement, it will be evident that almost every individual undergoes different changes of posture and movement; and this implies that the exercises have to be adapted to every person individually. This may seem impracticable, but the difficulty is overcome by advising the patient to perform precisely those movements which are most irksome to him. There should be no undue forcing,

cauda equina with pareses and prolonged incontinence resisting therapy are liable to occur. The president of the Congress for Chiropractice at Freudenstadt (1956) emphasized this once again; he made a distinction between a minor protrusion, when manipulation is admissible, and a manifest hernia nuclei pulposi, when it was useless and contraindicated on account of the great risks entailed.

Thus the problems surrounding this method of treatment run in circles. On the one hand, a single treatment may have resounding success; the treatment is useless for hernia and is even dangerous. Yet in practice, hernia nuclei pulposi can apparently only be diagnosed with probability on clinical grounds. The question therefore remains open as to when chiropractice should or may be prescribed for low back pain.

One cannot help but be struck by the following contradiction: Success is claimed to attend one manipulation, or at most once repeated. If, therefore, it happens *not* to be successful, what useful purpose can be served by undergoing these manipulations regularly for months on end, as patients coming to us with persistent symptoms have done over and over again?

5. A few words are in their proper place here about the *methods by which the patient is stretched*. As long as the patient's subjective intimation is taken as a measure of the force with which stretching can be applied, this simple procedure may be helpful without entailing much risk. Several patients found that their symptoms remitted when the body hung from the arms and so forth, using ordinary gymnasium equipment. But complicated frames have been contrived on which the anaesthetized patient is stretched. Even manual treatment under an anaesthetic is potentially dangerous, in that it may induce irreparable lesions of the cauda equina. We know of some such cases. Krayenbühl (p. 56), Armstrong (p. 177) and several other authors have mentioned it.

6. Sometimes a patient will ask about the therapeutic value of *medicinal baths, Kneipp cures and so forth*. As a rule, neurosurgeons, neurologists and orthopaedic surgeons have little practical knowledge of these and other natural curative treatments. The professional literature seldom has much to say about the scientific basis for such treatment (*e.g.*, sulphur baths being alleged to check the breakdown of hyaluronic acid in cartilage). Kusche (Freudenstadt, 1956) suggested that natural medical practice, chiropractice and neural therapy had gained a footing because the old-fashioned specialist disciplines (internal diseases, gynaecology, surgery, neurosurgery) had signally failed to discover really effective treatment for low back pain. Judging by what patients said, it would seem that natural medical treatment acted as a psychic and physical tonic; many neurasthenic complaints vanish after a few months of moderate

It should be added that the cost involved in the treatment is prohibitive and out of all proportion to the practical benefit the patient can hope to derive from it

3. *Neural therapy* consists in the acupuncture of, or injection of a few drops of Procaine (or Impletol) into, certain parts of the body which are considered to be foci of a neural disturbance. These parts are found empirically, the quest being guided by the experience of the therapist. A few seconds after the injection, pain or other symptoms may disappear, from other parts of the body as well ("Sekundenphenomen"). This is our personal point of view: we believe that it is possible to influence symptoms by neural therapy; we have seen it demonstrated by Hünecke, Düsseldorf, and had similar findings, ourselves. But we consider that the patients qualifying for this treatment are far fewer in number than the neural therapist appears to think (see p. 361).

The effect of this treatment on backache is disappointing (Zukschwerdt, p. 156). Epidural or periradicular infiltrations or paravertebral anaesthetization of the nerve outside the intervertebral foramen are not included, as the "Sekundenphenomen" is lacking Hunecke, who is perhaps more optimistic about the efficacy of neural therapy for low back pain generally than we are, has no illusions as to its power over hernia nuclei pulposi This has been verified in practice. Indeed, it is difficult to conceive how a mechanical factor could be influenced by such means.

4. *The chiropractor* manipulates with the object of rectifying the relative positions of various parts of the spine. We can well imagine that mechanical deformities would respond to that treatment and, in fact, patients relate how they have again been able to move without pain after being manipulated. But, as soon as it is claimed that a number of internal diseases can be cured by manual repositions on the spine, this therapeutic venture becomes shrouded in mystery. Zukschwerdt, who admits to his faith in the value of chiropractice and gives several important hints in his book as to suitable manipulations, writes (p. 226): "Hitherto no statistical, anatomical or experimental proof has been adduced of the existence of a disposition to diseases brought about by compression of vegetative fibres in the intervertebral foramen. And that leaves the major part of chiropractice unproved".

Failures as well as successes of chiropractical treatment are reported. Some patients were admitted to our clinic for surgical treatment after suffering serious neural loss in the nervous system following chiropractice applied elsewhere. This danger has been widely publicised in the literature, *e.g.*, Brussatis; Ford and Clark (see Spiegel, 1957, p. 144); Kostić (p. 76); Krayenbuhl (p. 56); Lewin (p. 128), Thurel (p. 25); Zukschwerdt (p. 160). Chiropractical manipulations are to be eschewed especially if a hernia nuclei pulposi is suspected, above all at the level of the L.V – S.I disc, when lesions of the

very successful specialists, but mainly of general practitioners with an inferiority complex. After following a short course, they see their way to acquiring a reputation as a "doctor with a special method of treatment" (bone setters), by virtue of which they too become specialists in a sense. They choose these mysterious fringing fields of medicine precisely because their lack of proficiency in the more reputable specialties will not be betrayed there. Although it is supposed to be an understood thing that

blood sedimentation rate test of the erythrocytes made, is, accordingly, a *testimonium paupertatis*.

The monomania of therapeutists without diagnostics derives from over-compensation for incompetence. This explains their defensive attitude to the outside world; their very terminology betrays antagonism to those who, endowed with critical insight and broader knowledge, fail to share their views. They do not refer to them as "people with another point of view", but as "opponents".

It is not surprising to learn that chiropractice, neural therapy, cellular therapy and natural medicine go hand in hand, because both the doctors and patients who are drawn to them are impelled by an urge towards the mysterious and the inexplicable. Both feel challenged by the unpredictable chance of startling success.

That there has been a renascence of these miraculous therapies in German-speaking areas, where mysticism has always been a strong element, is not astonishing

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to

(and sometimes vice versa), the contents are often difficult for the doctor to understand and the meaning, it seems, deliberately obscured.

Let us recapitulate briefly:

The therapeutic effect of physiotherapy and natural medical treatment is spoken of in fairly appreciative terms.

As far as is known, the application of neural therapy does not involve any danger. In selected cases there would not seem to be any reasons against trying this method when other treatment fails. It should, however, be conditional upon proper diagnosis being made and it should not be unnecessarily prolonged to the exclusion of, possibly, more effectual treatment. In the presence of a radicular syndrome presumed to be caused mechanically, such as that resulting from a hernia nuclei pulposi, the method is of no avail.

As far as chiropractice is concerned, the following are contraindications for manipulations to the spine

True dislocations and fractures,

Increased B S R.

Backache and sciatica combined with sensory disturbances, reflex changes, pareses, atrophies or micturition disturbances.

Very prolonged treatment for spondylolisthesis.

Bechterew's disease.

According to Brussatis, when these contraindications were respected, treatment produced the following results in sufferers from backache with radicular pain:

rest in beautiful surroundings, a light diet and the assistance of physiotherapy. Meanwhile, any serious disorders there may be are discovered by the supervising doctors. At the end of the cure there remains a residue of patients with persistent subclinical symptoms and strange, puzzling complaints. This is where the neural therapist and chiropractor feel at home. The patient has acquired the virtue of patience and the illness is not of a serious nature. Thus conditioned, persons within this group are the ideal patients to submit to a prolonged, somewhat mysterious treatment with an array of quite different medicines and so forth from those "all the other doctors had tried out in vain".

In view of the general interest evinced among doctors in this subject, we decided also to study it in practice. Just because, as stated several times, we are convinced of the therapeutic [illegible]
and if specially [illegible]
moreover, from [illegible]
practice. We als[illegible]
by the president of the Congress for Chiropractice (Freudenstadt, 1956). It should be emphasized here that our views rest on experience in chiropractice gained on the Continent of Europe only. Mulder (1958, p. 1165) has the temerity to assert that in Europe the majority of chiropractors still stand at the level of ordinary quacks. In our opinion a distinction has definitely to be made between those who can legitimately be so qualified and American chiropractors, for instance, who pass through a rigorous course of four years in diagnostics and therapy.

The careful enquirer will find that it is not only certain patients who fall into a group of persons with a preference for mysterious forms of therapy; doctors who feel drawn to practising unconventional methods likewise fall into groups.

First we have the *qualified expert* who, grounded in general medical knowledge, masters the diagnostics of this specialism he has chosen and has experience in it These colleagues do not resort to neural-therapeutic and chiropractical measures until they have performed a thorough preliminary clinical examination

Secondly, there is the *specialist* who, although he is an experienced practitioner in his own subject, taking advantage of the reputation he has acquired, experiments in an inexperienced manner on the borderline of neural therapy and chiropractice. Among this group are also to be found those who are *critically interested, i.e*, those who, relying on their practical knowledge of their own specialism, are always on the look-out for new ways and means and wish to be acquainted, though critically, with the new trends

Lastly, we have the *uncritical ones* who, with little knowledge, become wildly enthusiastic about events which they do not understand. To them, the simplest truths, which appeared years ago in the literature, are a revelation; nor do they realise that many manipulations and activities which they call neural therapy and chiropractice have long been part and parcel of the orthopaedic surgeon's, the general surgeon's, the neurologist's and the physiotherapist's therapeutic arsenal.

What lies at the root of the short-sighted mania for investing a simple local treatment with mystical powers to conquer pathological processes anywhere in the body? With exceptions, of course, this group of persons turns out to consist of a few not

up by giving him gymnastic exercises to do and to allow him to take up sport. It is better for the asthenic to do physical exercises and continue to work, provided it be not excessively hard, than to wear a corset; the former will put him into relatively optimum condition, whereas immobilisation of the spine by wearing a corset only leads to weakening of the muscles. The hard-driven housewife has neither time nor energy to spare and this perhaps also applies to the manager of a business and to so many others engaged in the intellectual professions. For all that, one should try to make it plain to them that ultimately they will be more useful to their households or professions if they avoid a psychic and physical breakdown. These people may be firmly convinced that they are merely doing their duty by putting every ounce of themselves into a heavily laden daily task, whereas in fact they are hastening towards a breakdown and, maybe, chronic ill-health. It is a frightening fact that this group contains a very large number of people who, on account of their exhausting daily task, have had no time since the age of 30 to 40 for mental or physical distraction. Yet this is very necessary with increasing years, even though prophylaxis will become gradually and progressively concerned with the prevention of diseases normally attending old age. The dangers of chronic inactivity in the aged (Hill, p. 6732) call for counter-measures, including active movement, especially in view of the greatly increasing longevity of men and women today. In "The Medical Aspects of Growing Old", Todd (p. 67) stresses the value and necessity of active physical exercise in advanced age.

Little thought is usually given to *sleeping habits* and the quality of mattresses until the first attack of low back pain. To our mind it is a mistake to advise anyone showing signs of incipient arthrosis to have long nights and to lie down in the afternoon. Female patients above the age of 40, above all, are given to this and, on the plea of a weak back, are all too ready to believe that they cannot do without it. If afternoon rest is necessary at all, it should be deducted from the night sleep. And all lengthy lounging on sofas and in armchairs is anathema. The "experienced" arthrotic sufferer knows better; he prefers a straight-backed chair. Is it unbounded energy, a wish to enjoy every minute of the time left to them, or their joints which have painfully stiffened up during the night, that drives many people of advanced age from their beds in the morning?

It is difficult to give any specific advice as to the *mattress* that should be used, because there are so many aspects to this question (Bungenberg de Jong, p. 912) The patient's financial means are also involved in it. It would seem to be healthier to sleep on a hard bed, but we are by no means sure that it should be boards, even for the therapeutic rest-cure. Any mattress on which

	Satisfactory result
Stretching followed by the wearing of a lumbar corset	78%
Stretching plus chiropractice	64%
Chiropractice followed by the wearing of a corset	65%

Mean satisfactory result	70%
Persons untreated	15%
Patients operated for diagnosed hernia	15%
	100%

These figures show that the above less usual methods of treatment, if applied by those qualified to do so, are also able to be effective.

A medical quack could be defined as a "therapist" with insufficient knowledge of diagnostics and therapy. It is they who, through their irresponsibility, bid fair to bring methods of treatment into disrepute which, in themselves, may prove to be valuable.

Prophylaxis

Care of the patient does not abruptly cease after he has been treated for the acute exacerbation of low back pain. Weizsacker (pp. 11 and 122) stresses the necessity of continuing to care for the patient's body and mind after an acute lesion has "healed". This applies equally to the conservatively treated patient. Sometimes nothing further can be done than to give him sound advice. But enlightenment is a primary necessity. The patient must be made to realise that it was his condition which brought on the attack and that the same predisposing condition persists, despite relief from the acute symptoms.

While it is right to put the facts before him, he should not be made unduly anxious or to feel that a relapse is inevitable; for this can be prevented by taking timely precautions. The patient must realise that the exertions required by the daily grind are no greater than what is strictly necessary, as those who are practised in their job do not need to make a call upon extra effort. This applies to manual workers and housewives and certainly not least to persons engaged in the lighter forms of physical labour. The patient should know that he is liable to be the prey to prematurely encroaching ageing and that it is still up to him to resist the process.

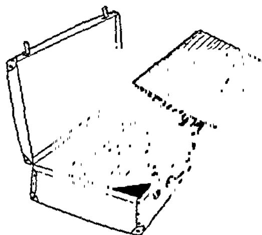
Prophylactic treatment should be a continuation of the conservative therapy. Recurrency should be *prevented by training; the patient should be shaken out* of his inertia; reserves of strength should be built up for any contingency. The young man with a spine bearing signs of degeneration will have to keep out of the way of excessively strenuous manual work and, perhaps, seek some other kind of employment. It is in no way inconsistent with this to build him

up by giving him gymnastic exercises to do and to allow him to take up sport. It is better for the asthenic to do physical exercises and continue to work, provided it be not excessively hard, than to wear a corset; the former will put him into relatively optimum condition, whereas immobilisation of the spine by wearing a corset only leads to weakening of the muscles. The hard-driven housewife has neither time nor energy to spare and this perhaps also applies to the manager of a business and to so many others engaged in the intellectual professions. For all that, one should try to make it plain to them that ultimately they will be more useful to their households or professions if they avoid a psychic and physical breakdown. These people may be firmly convinced that they are merely doing their duty by putting every ounce of themselves into a heavily laden daily task, whereas in fact they are hastening towards a breakdown and, maybe, chronic ill-health. It is a frightening fact that this group contains a very large number of people who, on account of their exhausting daily task, have had no time since the age of 30 to 40 for mental or physical distraction. Yet this is very necessary with increasing years, even though prophylaxis will become gradually and progressively concerned with the prevention of diseases normally attending old age. The dangers of chronic inactivity in the aged (Hill, p. 6732) call for counter-measures, including active movement, especially in view of the greatly increasing longevity of men and women today. In "The Medical Aspects of Growing Old", Todd (p. 67) stresses the value and necessity of active physical exercise in advanced age.

Little thought is usually given to *sleeping habits* and the quality of mattresses until the first attack of low back pain. To our mind it is a mistake to advise anyone showing signs of incipient arthrosis to have long nights and to lie down in the afternoon. Female patients above the age of 40, above all, are given to this and, on the plea of a weak back, are all too ready to believe that they cannot do without it. If afternoon rest is necessary at all, it should be deducted from the night sleep. And all lengthy lounging on sofas and in armchairs is anathema. The "experienced" arthrotic sufferer knows better; he prefers a straight-backed chair. Is it unbounded energy, a wish to enjoy every minute of the time left to them, or their joints which have painfully stiffened up during the night, that drives many people of advanced age from their beds in the morning?

It is difficult to give any specific advice as to the *mattress* that should be used, because there are so many aspects to this question (Bungenberg de Jong, p. 912). The patient's financial means are also involved in it. It would seem to be healthier to sleep on a hard bed, but we are by no means sure that it should be boards, even for the therapeutic rest-cure. Any mattress on which

the body can lie fully outstretched without any sagging of the spine seems suitable to us. Any mattress that sags is unsuitable. People who travel much may suffer considerably from having to re-accustom themselves over and over again to a different bed. They can help themselves by taking along with them hinged, foldable boards of three-ply wood which they can pack in their cases. It appears that most sufferers from chronic backache do best with their thin three-ply board between a box mattress and a Pullman mattress in good condition. The arthrotic sensitive to cold, however, feels a draught from the separately sprung Pullman compartments owing to air circulating when he moves. The foam rubber mattress has the same drawback. For these people the ideal seems to be a foam-plastic mattress separated from the underlying box matress by a three-ply board, this combination giving the warmth of a featherbed, the flexibility of the Pullman and the inflexibility of the board.



A psychiatrist should be called in for *psycho-therapy* and to decide whether *it is indicated. Sympathetic human approach by a doctor not trained in psychiatry* will suffice to help many patients, however. Every practitioner is confronted with this, because spinal symptoms are so often complicated by psychological aspects. In fact, it is his duty to tackle this problem if the prejudiced patient flatly refuses to see a psychiatrist. It is psychologically natural that a bond should come to exist between the surgically treated patient and the surgeon who has plunged the scalpel into him; on the one hand this lays an obligation on the surgeon to make good use of this bond in post-operative treatment, as his suggestions will then be gratefully accepted. The relation may become troublesome, on the other hand, if the patient takes to consulting "his" surgeon for quite different complaints and, in spite of advice to the contrary, refuses to go back for after-treatment and follow-up to the doctor who first saw him

The indication for surgical treatment of an organic disorder in a patient who is at the same time psychically disturbed in a pronounced degree raises serious difficulties. The dilemma is this. Is it to be expected that the psychic

disturbances, which will naturally persist after the operation, will elicit other pathological conditions through which the patient is no better off than he was before the elimination of that one organic lesion? Or is it to be supposed that the patient's psychic condition cannot be remedied as long as the organic abnormality persists, an abnormality in which obviously many of the disturbances to be psycho-therapeutically treated are rooted? The more serious the threat to the life or vitality of the patient this lesion is, the easier is the choice. Failing an absolute indication, it is our personal opinion that a serious lesion of this kind should only be attacked surgically if the psychiatrist considers the patient to be a promising subject for successful psycho-therapy in the post-operative period. We shall not discuss the criteria adopted (I.Q., age and so forth), nor the kind of psycho-therapeutic measures to be taken. It will be evident that analysis by depth psychology is a very different thing from making suggestions in conjunction with some form of physical post-treatment.

Surgical Treatment

Technique, anaesthetic, approach, wound management

We called conservative therapy the treatment of undiagnosed low back pain. The explorative character of surgical intervention, on the other hand, is usually helpful in disclosing the nature of the disorder. It is therefore easier to assess the therapeutic value of certain surgical interventions for given abnormalities.

When comparing the records in the literature, it is well to remember that results of surgical therapy almost invariably refer to treatment for hernia nuclei pulposi. Details of post-operative follow-up are mostly accompanied by the description of a special operating technique favoured by the author. This also makes comparison between the results of surgical therapy extremely difficult.

Authors differ widely in their views as to indications for a particular method of operation and after-treatment, yet the difference in their views is not reflected in the operative results After making several attempts to set out these different views in groups, we have decided, after mature consideration, to refrain from doing so. Instead, we shall merely record our own experience, only very occasionally appealing to comparable material in the literature.

Laminectomy, for the removal of hernia nuclei pulposi, is described in numerous publications. For the purposes of this study we feel that it will suffice to give technical details involved in the operative treatment for the degenerative status of spinal disorders Of these, hernia is only one aspect; and, where a herniatomy is concerned, we shall describe only those steps in

detail which, after the simple extirpation of the protruding nucleus, should to our mind be taken to combat or limit the remaining general consequences of the degenerative status.

Biemond (p. 243) says that, in his opinion, herniatomy should be performed by a neurosurgeon. The underlying idea could be differently formulated, *viz.*, "Operations on the nervous system should only be performed by surgeons thoroughly experienced by training and practice in that subject". So long as there is no special discipline for the surgery of the spine and cauda equina, the neurosurgeon is the only surgeon thus qualified. If this applies to the surgical treatment of hernia nuclei pulposi, the same cannot be said of operations for spinal disorders in a general sense. Depending on the clinical picture and associated indication, it is a defensible standpoint that any experienced surgeon dedicated to the treatment of spinal complaints should be competent to perform them. The crucial point is that the contingency of any necessary manipulation of a nerve must then be ruled out before the operation by the verdict of the consultant; and it seems to us that a neurologist will very rarely be able to give such an assurance with certainty. Therefore, the safest course is to entrust the *operation to a surgeon qualified and skilled in both.*

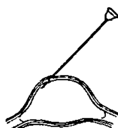
In what follows it cannot be constantly repeated that every case in every 100 patients in which failure is converted into success by special care increases the number of successful operations by 1% in the statistics. It is self-evident, but to realise it makes up for all the extra trouble and care given to each and every patient to make sure that everything is satisfactory. We say this, hoping that it may be accepted in extenuation of what may be felt as rather excessive insistence on detail in the pages to come.

There are some surgeons who perform a laminectomy, for whatever purpose, with the patient under a general anaesthetic, whereas others always administer a local or spinal analgesic (Spurling, p. 94). We do not find that there is any hard-and-fast rule for this. Some patients' nervousness and, especially, vaso-lability call for general anaesthesia. This, however, stands at a great disadvantage compared to a local anaesthetic when, by active co-operation, the patient is capable of assisting the surgeon to correct the level diagnosis to the last decimal, so to speak. The patient's reaction to monoradicular stimulation affords a clue to the direction in which the seat of the trouble is to be sought. In view of the shortcomings of the diagnosis, this added evidence is quite often decisive for the result. It is of even greater value in a so-called negative exploration for hernia nuclei pulposi, as in that event an attempt may be made to combat the radicular abnormalities by performing a guided selective sensory rhizotomy.

In our opinion, local analgesia (with 1% Procaine hydrochloride) is almost invariably to be preferred. Faced with ill-defined syndromes, we nearly always refused to undertake the operation if the patient made it a condition that he should be put under a general anaesthetic. In the past, we had occasionally capitulated, with the result that the negative findings and the impossibility of doing anything else to help the patient – by means, say, of a rhizotomy – decided us never to give in on this point again.

The so-called "overhaul operation" is usually performed on the elderly, when several levels are explored on both sides. Here the advantages of general anaesthesia are obvious, in view of the profuse haemorrhages to be expected from the osteoporotic bone and the distended epidural veins.

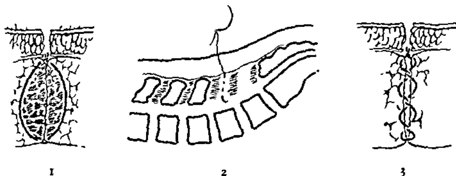
Manipulation of the root is often painful to patients being operated upon under a local anaesthetic. As against this, however, *Procaine* can be administered *intradurally* with perfect safety as soon as the abnormality has been located and the plan of procedure decided upon. We injected 5 c.c. of Procaine hydrochloride of 1%, from which the patients suffered no distress. Leakages can be prevented by introducing the needle tangentially, so that the punctures in the dura and arachnoid shall not be opposite each other. Nor is any harm done by anaesthetising a single root with 1 to 2 c.c. of Procaine.



We have all our patients *prone* for the operation, with exaggerated lumbar kyphosis. The objection sometimes voiced against this position is that it is apt to cause congestion of the vena cava, resulting in haemorrhages from the congested veins into the epidural space. In order to find out whether this really is so, we rectified the lumbar kyphosis when haemorrhages occurred in some patients, but did not find that it made much difference. We have seen operations on patients in the lateral position, but failed to see that there was less loss of blood than with the patient in the prone position, which is so much more convenient for the surgeon. In this respect, the knee-elbow position is by far the best, but is not practicable on account of the duration of the operation and the local anaesthesia which we consider necessary.

The question as to whether a laminectomy should be performed *via* a large wound or a small one has been the subject of some discussion (Mack, p. 469, Weddell *et al.* p. 178). The objection raised to a small wound is that, through traction of the retractors, the muscles become anaemic or the rami musculares are injured, causing secondary changes subsequently resulting in low back pain. The detection by electro-myelography of changes in the muscles does not, to our mind, provide a convincing argument, as they are also seen to take

place after laminectomies *via* large exposures. Nor is the criticism respecting the occurrence of low back pain valid, since the cases of it after 1000 laminectomies with no more than 7 cm exposed were not more numerous than in the statistics produced by authors favouring ample room for approach. The criticism is rebutted, we think, by the results in actual practice, seeing that the residual symptoms found at the post-operative check-up of hundreds of laminectomy patients who have been operated upon *via* an explicitly small exposure are not significantly more numerous. Lesion of the spinal muscles can be limited by not retracting them farther lateralwards than is strictly necessary. Moreover, it would seem to us reasonable to suppose that bundles of muscles detached for a short distance are less likely to be damaged than those thus treated for a greater one. Actually, one can see that the muscle retractors strain mainly against the long spinal fasciae and the extent to which the retractor can be opened depends on the prevailing tension here. We are convinced that the vast majority of post-operative muscle lesions are caused, not by the detachment, but by the suturing of the muscles. Many surgeons suture the spinal muscles deeply in such a way that ischaemia results (1).



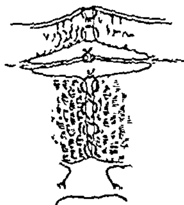
1 2 3

It has been our consistent practice to close the muscles with catgut, exerting no more traction than strictly necessary to maintain the opposed bundles in contact. The sutures are made preferably tangentially in the ligamentous thickening of the muscle fasciae where these had been attached in the midline *between* the spinous processes (2). The remaining openings between the muscle bundles are approximated one to another as shown in the illustration (3). This entails the risk of less effective staunching, just because the muscles are *not* constricted, but it is only a problematical risk and can be counteracted in the post-operative period by puncture. A small wound has, moreover, its *psychological advantages*. The patient has yet to be born who does not, as soon as he can, contrive somehow to get a glimpse of his wound after the operation. The neater and smaller it is, so much the easier is it to make light of the whole

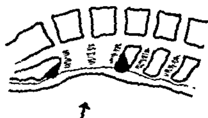
operation and it is an incentive to take up the exercises devised for his benefit with the least possible delay. The psychological effect is all the more important in patients who, through a trait of character, are abnormally preoccupied with their scar, so much so sometimes as to produce symptoms deriving from a psychological basis. The value then of an unobtrusive scar will be evident.

The importance of closing a wound cleanly and neatly becomes clear when one finds that praises of the scar go the round, from patient to patient, sometimes reaching the ears of sufferers from low back pain before the practitioner concerned has contemplated surgical intervention.

Before the skin incision is closed, we separate the subcutis from the muscle fascia. This prevents sclerotic processes through which formerly, when this precaution was not taken, the scar sometimes became attached to the ligament of a spinous process. The application of the edge of the wound is reinforced by a separate layer of fat sutures. The skin is joined by inverted subcutaneous skin stitches, making external stitching of the skin superfluous. Hence there is nothing to deform the scar, which heals well. These precautions might seem to be exaggerated, but the psychological effect upon the patient of speedy recovery with a small scar healing well is greater than might be supposed. In view of the many psychological aspects of low back pain, this is not surprising.



The adjacent spinous processes are nibbled off obliquely to prevent pressure from projecting parts of bone upon the skin in the supine position.



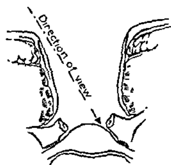
Some surgeons hold that the least possible amount of bone should be removed for a herniatomy. Their procedure is to nibble away only the lower margin of the arch involved and to excise the ligamentum flavum. It is difficult to understand how such an exposure can afford the surgeon a clear view. Possibly this approach suffices for the extraction of a detached hernia or the excision of a disc protrusion. This standpoint taken by a surgeon is defensible if he is satisfied with 60% to 70% successful operations, when allowance will have to be made for the possibility of having to perform a second big operation if symptoms persist. We do not know to what extent persisting symptoms



cleared up after second operations of this kind. Persuaded as we are that the patient stands to gain by the best chances of the first laminectomy, we do not feel attracted to the above point of view.



Other surgeons confine themselves to a so-called median approach. They nibble away the central part of an arch, often reported as "removal of the whole arch". We have had ample opportunities in the past of re-exploring such cases and it seemed to us that this approach fails to provide an adequate view of the field of operation in the epidural space. After all, the pathology of low back pain takes place in the lateral recess. To reach this by means of a median approach (when the lateral parts of the arch remain), there is no alternative but to explore obliquely. This means keeping the roots more towards the middle than would otherwise be necessary and, in order to see properly from a slanting lateral direction, a large, widely retracted muscle wound has to be made. Upon enquiry we found, indeed, that the same surgeons who favour this approach are advocates of the large wound and are against distancing the muscles with retractors.



Like Lewin (p. 41), we consider the main function of the arches to be to shield the vertebral canal posteriorly and thus protect the nerve tissue. The supporting function of the arches is far less important. Accordingly, we do not think the removal of one arch detracts from the stability of the spinal column. We are supported in this opinion by the post-operative results of the many cases in which this was done and the results of which were better rather than worse than those generally reported in the literature on herniatomy.

For a simple herniatomy at one of the lowest lumbar levels, it is a matter of routine with us to *remove the L.V arch entirely*; *i.e.*, we nibble off the arch in a lateral direction far enough for the intervertebral foramen involved to be accessible almost *vertically*. As a whole, this procedure has the following advantages

A small exposure suffices.

The muscles do not have to be stretched to the utmost.

The ligamentum flavum is excised completely; there are no shrivelled remains in the lateral recess.

The roots need to be held less medially.

Free view of the lateral part of the disc.

Free view of the intervertebral foramen.

Easy approach for foraminotomy.

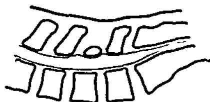
Chance to explore for anomalies.

Two levels in full view.

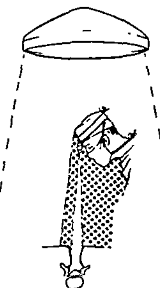
At the end of the operation there is room to spare, which is a preventive against certain complications.

The decompressive effect of the laminectomy is thereby enhanced.

Exploration on both sides is facilitated.



If the arch were left intact and two levels were nevertheless explored, the dural sac would be seen slightly protruding through both approaches. This means that in the middle the arch remaining *in situ* indents the dural sac as it were. We do not believe that this compresses the intradural roots; but the arch is then a predisposing factor to stenosis locally. Any adhesion, granulation and so forth, due to blood left behind or to whatever cause, will have a better hold and exert more compression upon the site of the dural sac's constriction.



The field of operation can be illuminated more satisfactorily if one arch is eliminated completely to just above the foramen. The light of the lamp falls almost vertically into the lateral recess; as the surgeon's line of vision is likewise directed almost vertically, his head intercepts the light from the lamp. Some surgeons who share this preference for the small exposure (Spurling, p. 95, *i.e.*, 3 inches) use spotlights. It is our custom to wear a head-lamp for every laminectomy, the only drawback being that, once used to this valuable facility, it is very hard to do without it.

The need for staunching the blood to the utmost is stressed by several authors (Spiegel, 1957, p. 327), in order to avoid complications. But the further one explores the lateral recess, precisely where the epidural veins are most strongly developed, the more probably will haemorrhages occur.

Osteoporotic bone is often found in the elderly. It is then necessary to cement the chiselled, wide-meshed bony tissue with wax. The vertebral

emissaries may be opened while posterior lipping is being chipped away, when any haemorrhages proceeding therefrom can (only) be stopped by swabbing the emissaries with muscle tissue. The walls of the veins in the epidural space are often pathological and do not shrivel up; instead, holes are burned into them upon coagulation. Again, the course is to stop these haemorrhages by swabbing.

However carefully the blood is staunched, some blood will collect in the deepest parts of the field of operation after loosening the retractors and while suturing the muscles. For all that, the prognosis for these patients is not *strikingly unfavourable. We are not so certain, therefore, that a little blood left* behind seriously increases the risk of complications. We did not consider it necessary to *drain* laminectomy wounds, except after sanguineous re-laminectomies. It is our common practice to make a puncture on the fourth day and, occasionally, once again later on. We do not consider that the necessity of drawing off about 20 c.c. of *sanguinolent fluid* at any one time points to a complication. This fluid, probably supplemented with Procaine from the muscles and, maybe, spinal fluid from leaking lumbar puncture perforations, collects under the detached subcutis. After the patient's mobilisation, moisture deriving from between the muscles is sometimes worked outwards and again fills up the subcutaneous space. We do not consider that this calls for re-immobilisation of the patient; quite the contrary; we think it is a good thing if the patient, by his own movements, works residual engorged serous fluid away from the muscles. Provided the necessary precautions be taken, secondary infections of the wound do not, in our experience, occur as the result of puncturing as a routine measure.

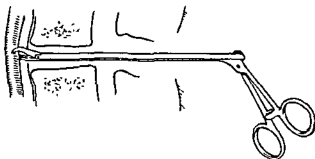
We have reason to believe that the post-operative engorged serous fluid is *supplemented by spinal fluid, yet that this does not necessarily imply that there* is a spinal fluid fistula. An ample exploration involves the re-opening of a lumbar puncture in the dura. Also, the covered opening one level above the field of operation may temporarily begin to leak. That is why we prefer the *lumbar puncture to be at the level of L.IV - L.V; steps can then be taken* during the laminectomy if fluid should issue from the dura opening, which is visible and can be sealed with a single tangential dura suture. To a certain extent the same risk is incurred with an intradural Procaine application. It is for this reason that we advocate the smallest possible needle, which should pierce the dura tangentially.

Ever since the various layers of laminectomy wounds have been generously supplied with antibiotics, in the form of Globenicol, wound infection has become a thing of the past. Sperl (see Spiegel, 1957, p. 325) reports in a like

sense. Laminectomy is a major operation. In the past, the complications resulting from an infected laminectomy wound were apt to be serious. The patient's well-being is at stake and that is why we consider the local use of antibiotics as a routine precaution to be justified, all the more so because the proximity of the lumbar region to the anus makes infection a risk to be reckoned with.

The mortality after laminectomy for uncomplicated spinal disorders, without counting tumours and the like, is exceedingly low. One of our patients died of general marasmus during the late war. His physical condition was so bad that, looking back, we realise that we ought not to have operated upon him, notwithstanding the severity of his symptoms. Actually this patient died four months after the laminectomy under circumstances which suggest the presence of a further complication. There was no post mortem examination. Apart from this single case, none of our patients died from the operation. Other surgeons report fatalities as the result of cardiac and vascular complications. Even including this kind of complications, the mortality is estimated to be less than 1% (Spurling, 1958, p. 115: 0.4%).

If the anterior aspect of the disc is pierced, there is danger of *injury to the major abdominal blood vessels* (Mack, see Spiegel, 1957, p. 328). Once in our series it was found that a chronic hernia nuclei pulposi had not only broken through dorsally, but had also perforated the annulus fibrosus ventrally. After the nucleus had been extracted, we were able to explore the ventral opening. At



first no symptoms were presented, but 24 hours after the operation the patient complained of abdominal pain. There were symptoms of irritation of the plexus and tenderness on pressure of Douglas's pouch. It was assumed that sero-sanguineous fluid, passing through this opening, had got behind the peritoneum. The post-operative course was complicated for some weeks by these symptoms and the patient subsequently retained minor chronic abdominal complaints.

Exploration of the lateral recess may be unexpectedly associated with

considerable loss of blood. Therefore, every patient is given an *intravenous infusion of saline*. Its great merits are that it keeps the patient in the best possible condition from the outset and affords constant contact with the venous system, so that emergency measures can be taken at once in the event of severe haemorrhage. Moreover, the patient suffers far less from thirst during the first few days after the operation. It also holds off nausea. The patient is allowed to drink immediately after the operation, coffee or a small quantity of alcoholic liquor. As our post-treatment *in toto* aims at starting on rehabilitation within a few days, such concessions serve to bring home to the patient the truth of our assertion that the operation "was not as serious as all that" and that he is quite able to follow out the instructions, which will soon put him on his feet again.

Despite reports to the contrary, the number of laminectomies performed for radicular pain and low back pain is not on the decrease. The vast majority of statistics refer to the treatment of hernia nuclei pulposi. We know for sure that the number of operations performed in the Netherlands on insured panel patients from 1952 to 1955 inclusive increased by approximately 10%. These figures, coming from the Central Office for Statistics at Utrecht, are not complete; they do not take account of the increase in population, which is less than 10%. The experience gained in the surgical treatment of hernia nuclei pulposi has refined the indications for it. If we now hear that, this notwithstanding, more hernias are being surgically treated, the only inference to be drawn is that an increasing number of well-diagnosed cases qualify for operation. A recent study of our personal material revealed that, relatively speaking, the number of high lumbar hernias is increasing, as is also the age of the patient earmarked for operation. This might point to refinement of diagnosis. As low back pain becomes better understood, more and more people appear to qualify for surgical treatment who were formerly considered to be inoperable. Hitherto we have very sparingly recommended laminectomy for patients *not* suspected of a hernia nuclei pulposi, but whose symptoms pointed to a degenerative condition of the back; sometimes we were able to relieve them, too. Possibly in future, as our experience of this ailment grows, this group will swell the ranks of surgically treated sufferers from low back pain.

Surveying surgical treatment, Goinard (p. 155) speaks of "des séries scandaleusement nombreuses". Such censure is merited only if the result does not, broadly, vindicate the indication as defined. It does not speak well for the medical organisation under certain circumstances in a given country if the indications for a form of therapy approved in other countries are held in very low regard. It could be countered that formerly, owing to lack of qualified, neurologically trained examiners, the majority of cases of hernia nuclei pulposi

were overlooked. In Portugal, where, we are informed, there used to be a shortage of neurologists, only six cases of hernia nuclei pulposi were surgically treated before 1946, though it had the same population as that of a country where more than a thousand cases had already been operated upon at that time to reasonably good effect or even to good effect. It was a comparison of that kind which brought us to realise, even in those early days, that the problems surrounding low back pain were dominated primarily by its diagnosis and formulation of the indications, rather than by the technique of operation.

Indication For Surgical Treatment

There is a tendency only to contemplate surgery when conservative treatment has failed. Opinions differ on this (Bonola, p. 476, Burns and Young, p. 245, Kostić, p. 76, Kemp, pp. 3054 and 3116, Milikan, see Spiegel, 1955, p. 344, Reischauer, p. 12, Shiners and Hamby, p. 450, Voris, p. 24, Zukschwerdt, p. 161, etc.). One author insists on a full year's conservative treatment, while another extols the results of an operation during the first acute stage of a radicular syndrome. Even where an uncomplicated hernia is concerned, the indication for herniatomy cannot be tied to the simple general guiding rules. The following facts have to be considered:

The gravity of the signs and symptoms

Severe pain without neurological symptoms is *not* a case for urgency, as it can be controlled by analgesics.

Micturition disturbances, pareses and saddle anaesthesia provide indications for exploration.

Marked (and painful) postural anomalies, persisting in rest, contraindicate continuation of bed rest.

The nature of the ailment

Exploration should be considered if ill-defined radicular symptoms associated with a manifestly degenerative back persist, despite continuous conservative treatment for at least one year, or recur chronically and provided they threaten the patient with invalidism.

The middle-aged sufferer from spondylarthrosis, having had several attacks of lumbago or sciatica in the past, is to be suspected of nursing the residual condition of a so-called "healed" disc lesion, possibly at several levels and accompanied by posterior lipping or narrowing of the foramen. An "overhaul exploration" could be undertaken if the patient's general condition is satisfactory and if he is threatened with invalidism.

considerable loss of blood. Therefore, every patient is given an *intravenous infusion of saline.* Its great merits are that it keeps the patient in the best possible condition from the outset and affords constant contact with the venous system, so that emergency measures can be taken at once in the event of severe haemorrhage. Moreover, the patient suffers far less from thirst during the first few days after the operation. It also holds off nausea. The patient is allowed to drink immediately after the operation, coffee or a small quantity of alcoholic liquor. As our post-treatment *in toto* aims at starting on rehabilitation within a few days, such concessions serve to bring home to the patient the truth of our assertion that the operation "was not as serious as all that" and that he is quite able to follow out the instructions, which will soon put him on his feet again.

Despite reports to the contrary, the number of laminectomies performed for radicular pain and low back pain is not on the decrease. The vast majority of statistics refer to the treatment of hernia nuclei pulposi. We know for sure that the number of operations performed in the Netherlands on insured panel patients from 1952 to 1955 inclusive increased by approximately 10%. These figures, coming from the Central Office for Statistics at Utrecht, are not complete; they do not take account of the increase in population, which is less than 10%. The experience gained in the surgical treatment of hernia nuclei pulposi has refined the indications for it. If we now hear that, this notwithstanding, more hernias are being surgically treated, the only inference to be drawn is that an increasing number of well-diagnosed cases qualify for operation. A recent study of our personal material revealed that, relatively speaking, the number of high lumbar hernias is increasing, as is also the age of the patient earmarked for operation. This might point to refinement of diagnosis As low back pain becomes better understood, more and more people appear to qualify for surgical treatment who were formerly considered to be inoperable. Hitherto we have very sparingly recommended laminectomy for patients *not* suspected of a hernia nuclei pulposi, but whose symptoms pointed to a degenerative condition of the back; sometimes we were able to relieve them, too. Possibly in future, as our experience of this ailment grows, this group will swell the ranks of surgically treated sufferers from low back pain.

Surveying surgical treatment, Goinard (p. 155) speaks of "des séries scandaleusement nombreuses". Such censure is merited only if the result does not, broadly, vindicate the indication as defined. It does not speak well for the medical organisation under certain circumstances in a given country if the indications for a form of therapy approved in other countries are held in very low regard. It could be countered that formerly, owing to lack of qualified, neurologically trained examiners, the majority of cases of hernia nuclei pulposi

would be medically incorrect to allow the patient to choose; it is a psychological mistake which the patient, unconsciously, never forgives. The medical practitioner cannot do more than put it to the patient in the light of his own considered view, and probably his representations will be tendered differently from individual to individual. In this way he stimulates the patient to express his wishes and the medical adviser takes the ultimate decision, making due allowance for those wishes.

In everyday practice, the indication for surgical treatment to combat pain seldom rests on medical grounds alone; the scales are tipped by the patient's longing to be rid of his distress, the sooner the better and, as he supposes, permanently. The indication is always difficult, particularly if it does not promise fully to satisfy the social demand for a *speedy* recovery. In our opinion, which is strengthened by the results obtained in following this course, there is practically no alternative, where sufferers from a manifest hernia syndrome between the ages of 20 and 45 are concerned, but to adhere to this rule: *If no marked improvement is seen to have taken place during trial mobilisation after two weeks' complete rest, the indication for surgical treatment on the basis of an extensive examination should be considered.* Continuation of the rest cure then offers little chance of complete recovery and is beset with difficulties, for which reason it is, to our mind, contraindicated, if only socially. Results have, moreover, shown that herniatomy offers the best prognosis if applied to young people for the removal of a hernia that has not existed too long.

The foregoing on the treatment of low back pain can be roughly illustrated by the chart on page 598.

Details of herniatomy

One method is merely to extract the protruding nucleus, but most surgeons excise the disc as completely as possible. If the longitudinal ligament has not been ruptured, a cruciate incision will enable it to be sutured later, but ample excision of the ligament is desirable if one wishes to remove the disc radically.

It is sometimes difficult to distinguish a physiological prominence of the disc from a prolapse. Occasionally one is tempted to leave a small prominence *in situ*, but in an explorative laminectomy, when the surgeon is looking for the cause of ill-defined symptoms, the tendency will be to see a prolapse in every prominence.

We were only able to discover by *transdural palpation* in 51% of our cases that the disc had undergone changes of a kind that demanded extirpation. In the remaining 49% the hernia was situated so laterally, or, no bigger than a

We consider that exploration is indicated for persistent violent pain despite long weeks of rest, even if the clinical symptoms do not wholly tally with the clinical picture of a hernia and a hernia cannot be demonstrated myelographically.

If *a person's movements suggest a disc lesion and there is also a radicular* syndrome, such as a hernia induces, other factors again enter into the indication from individual to individual:

Age. Extreme conservatism will be maintained towards young people (under 16) and surgery will be eschewed unless neural disturbances make it imperative.

Older people (above 50) are scarcely to be suspected of an uncomplicated hernia. If exploration is decided upon, the surgeon must be prepared to encounter spondylarthrotic changes in the epidural space which he will have to correct. The indication is that for "overhaul operation", which has yet to be described.

Duration of the symptoms. It makes all the difference if a person has already had several attacks of "sciatica". Even if there have been frequent remissions through rest in the, sometimes long, clinical history, meanwhile the condition of the spine, the muscle/ligament system and of the individual *in toto* has deteriorated. With fair probability the time can be seen approaching when the recurrent disc lesions and progressive spondylarthrosis will bring about so many complicating secondary changes, that the root system will be permanently compressed in spite of conservative precautions. Should it look as though a decompressive laminectomy will become inevitable sooner or later, it will be preferable for several reasons not to procrastinate until the patient is older still.

Apart from the psychic aspects adhering to the clinical picture, the *psyche of the patient*, especially his character and temperament, is an integrating factor helping the consultant to formulate the indications. It will soon be apparent whether a patient can bear prolonged conservative treatment and whether he is capable of co-operating to the full. It will tell, too, whether a patient wishes to undergo an operation or not, although, of course, the medical indication cannot be made to depend on this. Sometimes the patient's attitude in this respect provides a yardstick for the gravity of the complaints. Besides, nobody is better able than the patient to realise the social consequences which affect him personally. The indispensability of the mother of a family, the economic position of the breadwinner running his own business, etc., are matters to which the doctor, thinking along medical lines, is sometimes apt to give scant attention as he considers the case rather than the human being behind it. It


Small bud hernia under nerve-root.


Bud hernia, some time after decompression of the root, showing increased protrusion.

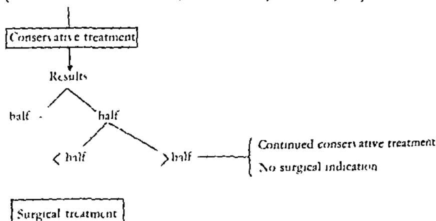
we judged it to be better to chisel out the ossified disc lesion, assuming that the low back pain derived from inadequate consolidation at the site.

The object of extirpating a protruding nucleus is to relieve the compressed root and to prevent further protrusion. For the latter the most effective course, we think, is to remove the nucleus and the major part of the disc as radically as possible.

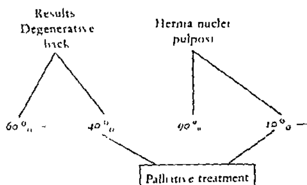
Many surgeons spoon out the intervertebral disc through the aperture where the hernia was excised. We doubt whether it is possible by this means also to remove the contralateral tissue completely. According to the records in the literature, contralateral radicular symptoms occurred after one-sided discectomy in about 10% of cases, a percentage that accords pretty well with our own findings in the period before we also systematically performed *contralateral discotomy*; thereafter it dropped to 2%. We do not mean to imply that these contralateral symptoms are the severe radicular symptoms which bear the stamp of a contralateral recurrency; they are, rather, vague, painful paraesthesias, distressing to the patient but for which few desired to be reoperated upon. We believe these residual complaints can be greatly reduced by

LOW BACK PAIN AND RADICULAR PAIN

(Unknown number → tumours, inflammations, deficiences, etc.)



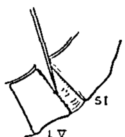
(depending on aetiology and condition)



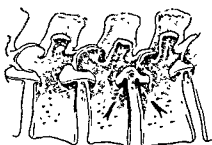
pin's head, under the root, that reliable information could not be obtained by digital palpation (bud hernia, see radiographs, p. 599).

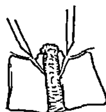
We have found no cases mentioned in the literature in which it was decided to leave a manifest disc hernia *in situ*. In two cases of spondylolisthesis of L.V relative to S.I we refrained from extirpating a considerable prolapse to avoid weakening the spinal column any further. The S.1 roots were decompressed. The patient was checked up for years and during that time the radicular symptoms did not recur, the patient retained the indeterminate backache that goes with spondylolisthesis. Similarly, we treated ossified, old disc lesions by decompression of the root running over them, effected by amplifying the lateral recess, foraminotomy and canalotomy. This we decided to do when the patient presented only radicular symptoms If, however, the low back pain dominated,

possibility of a post-operative motor disturbance through a combined neural loss of roots L.5 and L.4 cannot be ruled out.



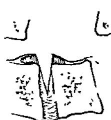
With the patient horizontally prone, the *L.V – S.I disc is slightly aslant the vertical.* If this is not borne in mind when it is about to be excised, the ventro-caudal portion of the nucleus and disc will be outside the plane of the excision. This portion may then be left behind and, after degenerating, will become detached and extruded.

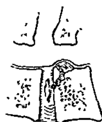




When a disc prolapses, the edge of the cartilaginous *laminae also emerges slightly*. After a discectomy one quite often sees the surrounding margins of the disc still protruding dorsally as far as the disc had initially done. Protuberances can then often be felt which turn out to consist of longitudinal ligament, fragments of nuclear tissue, of the annulus and of the lamina and curled margins of bone. The edges of the vertebra, where these fragments are found, are chipped off with chisel and hammer over the whole width, usually right into the foramina, as a matter of routine (see micro-radiographs on page 602).

Nuclear tissue may penetrate under the longitudinal ligament to a depth of 4 to 5 mm. This tissue *sometimes remains behind after excision of the disc if* search is not made for it. Nuclear tissue, which has thus found its way there, is frequently found in the material chipped off the vertebral margins and it can also be extruded from openings in the posterior aspect of the vertebral bodies. If the intervertebral disc from which this tissue presumably derives does not prolapse, we leave it intact.





Loose sequestra of nuclear tissue may be situated in the intervertebral canal

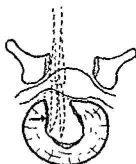
spooning out the disc contralaterally at the same time. In 30% of our cases we found a contralateral prolapse of the disc, though less pronounced than on the side where the initial trouble began; by way of exception, the contralateral prolapse was bigger even than on the painful side.

Although clearing the central part of the disc will reduce the tension of the remaining disc tissue, it is easy to see why conditions are comparatively favourable to a contralateral protrusion after a unilateral discotomy. The *annulus fibrosus may be compared to a ring holding the disc dorsally. One-sided* incision breaks its continuity. The intervertebral space is always liable to narrow after clearance of the disc and this brings pressure to bear upon any remaining disc tissue. If the incised annulus is no longer able to keep this tissue within the intervertebral space, it is more than likely to protrude. Residual nuclear or disc tissue, if detached or necrotic, will migrate to the homolateral space whence the nucleus was removed; but, if attached to some extent – as in older people – to the contralateral half of the annulus, it cannot move away and is extruded on the spot.





A further advantage of contralateral excision is the opportunity the approach affords for exploration and lateral decompression of the contralateral root system, combined with foraminotomy, etc.

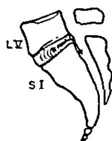


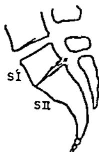
Lastly, surely it is to the good, as far as the stability of the spine is concerned, that the disc should be cleared symmetrically?

The intervertebral disc is oval and extends laterally to the incision to a considerable extent. These are points to be remembered in the homolateral removal of nuclear and disc tissue, particularly by the medial approach. This lateral part of the disc is more accessible by the lateral approach – chipping the pedicles laterally-wards – and fragments of disc tissue can therefore be spooned out from the side.

In the lateral excision of the L.IV – L.V disc there is a risk of *damaging the L.4 root*, which, unlike the L.5 and S.1 roots, is barely visible (as is also the associated ganglion), because it runs high (cranially) in its course through the foramen. Its presence is easily detected by electric stimulation. Damage may be inflicted if the scalpel is carried too far upwards. After intradural anaesthetization, the patient will be aware of little more than a muscular shock; *but if, pre-operatively, the function of the L.5 root was already reduced, the*

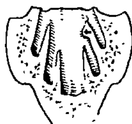
of the S.1 or S.2 root. Their presence can usually be deduced from certain facts of the clinical history.





During exploration along roots S.1 and S.2 we found nuclear tissue derived from a rudimentary S.I – S.II disc on three separate occasions.

When exploring along roots S.1 and S.2 one should remember that the cartilaginous or bony septum between these two canals may have thickened and may press on one of the two roots. Bony spur formation is described in the literature and we ourselves came across two examples of it.





Bony spur formation on the septa intercanalia S.I and S.II (verified at operation).



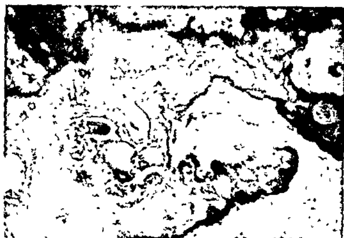
The surgeon is sometimes puzzled as to what to do with a "concealed disc", so called. First of all the roots, which not infrequently adhere to the longitudinal ligament in these places, have to be freed. Then, by careful exploration, it is necessary to establish whether there has been any extensive lipping of the vertebral bodies adjacent to a sunken disc of the kind (probably as a reaction to a disc lesion in the past). If the patient's radicular symptoms



Upper part of osteophyte with compact bone structure, covered with a thick layer of connective tissue (sensitive to pressure?).



Crushed vertebral margin; conglomeration of bony tissue, connective tissue with few cells, cartilage and some fatty tissue.

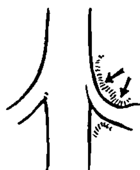


Growth of osteophyte; irregular formation of bone, connective tissue, cartilage and bone marrow.

it is a little hard to realise that more than 11 lb. of disc tissue were removed in these 700 herniatomies!)

Details of explorative laminectomy

After the arch between the two discs to be explored has been nibbled away, the epidural space is approached first, on the side opposite to that which has been giving the patient most trouble. This makes more room to explore the other side. When the arch has been removed, the ligamentum flavum is largely free. It is stripped as much as possible so that no severed parts shall remain behind. The epidural space is then systematically widened laterally by chipping the pedicles. This clears the lateral recess and the margins of the intervertebral foramina become visible. If the pedicles fan out, they have to be chiselled to open up the entrance to the foramen. The lateral insertion of the ligamentum flavum is removed together with the chippings of the pedicles. This is particularly necessary when the lumbar vertebral canal is wide, because the ligamentum flavum is then likewise very broad and is liable, after section, to contract into blobs of tissue of considerable size, which may compress the roots and give rise to adhesions. The epidural course of the roots and, in the majority of cases, the middle portion of the ganglia are now visible and can be explored for anomalies.



Ganglion compressed by posterior lipping.



Root stretched over a foraminal margin.

The course of the roots is carefully traced to see whether they run straight to their foramina or whether they are impeded in any way by the cranial



Bud hernia under nerve-root.

Opening burned into the longitudinal ligament.

In the same way that the protrusion of the bud hernia increases some time after decompression, the disc tissue herniates through the opening in the ligament.

predominated, we merely decompressed the roots by enlarging the lateral recess through foraminotomy and canalotomy. Only if the patient suffered from severe low back pain did we extirpate the "concealed disc" and then proceeded to chisel off the lipping down to the foramina on both sides.

While a disc is being explored, an *opening may happen to be burned into the longitudinal ligament* at the same time as the veins coagulate. Possibly this would not much affect a normal disc, but, if it already showed any tendency to protrude, we considered it necessary to extirpate it, even if we had not previously intended to do so. There is too great a risk of the disc tissue's emerging slightly through the opening in the ligament and, as pressure increases, actually herniating.

The herniatomies of which we shall presently give the results were performed along the lines described above. It was a matter of principle to carry out as *complete a double-sided discectomy as possible. Recurrent symptoms resulting* from protruding disc tissue left behind are thus prevented and we think the risk of post-operative symptoms is also lessened. There is room to spare for the roots to run freely and into which scar tissue can withdraw; and this, again, reduces the risk of root adhesion.

The average weight of the disc tissue extirpated on these basic principles was 9.88 grams (in 500 herniatomies)! It was 4.26 grams (in 200 herniatomies) when the previously usual method was employed, which, after all, was described at that time as a "complete discectomy"; but it did not entail all the above details. (Being accustomed to think in minute quantities per laminectomy,

has to be removed with a sharp chisel. This opens the foramen wider, the joint usually being saved. In 11 cases the joint was opened; of these, 7 patients were found to be symptomless at check-ups, one patient still had some slight low back pain and the results with 3 patients were unsatisfactory, but this does not mean to say that the symptoms of these 3 patients resulted from opening the joint.

The foraminotomy is completed by chipping off the vertebral lipping continuing into the foramen. Although this is not visible radiologically and the vertebral margin in the midline scarcely rises above the normal, marginal lipping may be so high in the foramen that it forms a gutter, as it were, around the root there. This is seen chiefly in the L.V – S.I foramen, hence relative to the L.5 root. Possibly the tendency towards this malformation is due to the position of the emissaries just above the lateral aspect of the lower margin of L.V (Schmorl, p. 18). Thus the vertebral margin offers less resistance to pressure, curls round and seals off the emissaries. The result may be congestion in the epidural space. Quite often there is haemorrhage from the opened emissaries while the raised vertebral margins are being chiselled. The gutter surrounding the root impedes its freedom of movement and causes a local stenosis. Even if the malformation does not itself cause compression of the root, under the existing conditions it would need very little further limitation of space to bring on symptoms. We call this abnormality the "*gutter syndrome*". The removal of this "gutter" was found to be beneficial to patients having no other disorders.

The overhaul operation

The complex of manipulations described above (clearing the lateral recess, canalotomy for the S.1 root, foraminotomy for roots L.5 (and L.4), chiselling lipping, chipping off spondylotic hooks in the foramina, removing the septum between S.1 and S.2, freeing the ganglia, and so forth) came to be known as an overhaul operation. Its object is to provide the lower part of the lumbar spine extensively with a new set of "bearings". Herniatomy is only part of the operation and is only resorted to if the findings make it necessary. The consequences of so extensive an operation should be well considered before the indications are formulated. The health and vitality of a patient getting on in years must be all that is to be desired. General anaesthesia and blood transfusion are necessary. As a rule, patients get through the operation well.

The place of predilection for local lumbar spondylarthrosis is, unlike that of hernia nuclei pulposi, around the L.III and L.IV vertebrae. That is why this level is included in a big overhaul operation. In view of the extensiveness of this surgery, it might be asked where one is to draw the line. Here it is necessary

margin of the foramen; if so, this margin is chipped or nibbled away. Sometimes the root is seen to run freely between the dural sac and the foramen and can easily be pushed up and down, but the ganglion lies immovably in the foramen. It is seldom possible to find out why this is so; occasionally one discovers that it is being held in place by lipping of the lower margin of the vertebral body. The ganglion becomes motile again when the foramen has been cleared and the bony margin chipped off.

It is not always possible to establish with certainty whether one of the above changes is the cause of the patient's syndrome. The above steps are taken as safety measures. The object of freeing the root system and the ganglia is mainly to prevent recurrence of the radicular symptoms. In special cases, *e.g.*, when syndromes indicate an overhaul operation, it is expected to produce an immediate therapeutic effect upon the existing symptoms.



Left Bony spur in foramen *Right:* Ventral margin of the superior articular process (dorsal roof of foramen) chiselled of in one piece.

Foraminotomy is a procedure introduced in the literature and differently described by various authors as Facetectomy (Ghormley), Intervertebral Foraminotomy (Briggs and Krause), Undermining Foraminotomy (Love). The dorsal boundary of the intervertebral canal – hence the site of the vertebral joints – is usually left intact The foramen is a flat oval in shape and is bordered on the dorsal side mainly by the superior articular process. After chipping away the fanwise extension of the pedicle, the ventral margin of the articular process

basis of exact numerical data. After changing a method that had not been very successful (*e.g.*, 30%), it would be easy, later, to prove the advantage of a new departure on the evidence of significant figures; but the herniatomy performed in the usual way had already shown a comparatively high rate of satisfactory results (80% to 85%); so the improvement gained by the supplementary intervention amounts to only a few per cent.

Re 2. An overhaul operation deals with the lower part of the lumbar spine. Since a patient's condition at a certain age depends on his constitution and precipitating factors, the indication for an overhaul operation cannot be tied down to any one particular disorder. The residual symptoms of a disc lesion, premature spondylarthrotic changes, anomalies of the nervous system and surrounding membranes; in brief, all the manifestations described above of the degenerative back may bring about a condition that calls for overhaul.

In the foregoing we have more than once stressed the fact that the object of surgical treatment is to clear up the symptoms, not to corroborate or rebut a diagnosis. It is immaterial to the patient whether he is being treated for this ailment or that, so long as he feels the better for it! For all practical purposes, herniatomy is still the most rewarding operation; by a surgical overhaul it may nevertheless also be possible to clear up complaints which were resistant to conservative treatment and used to appear to be incurable.

The prognosis of the degenerative or the deformed back is considerably less favourable than that of hernia, but, if properly indicated, the overhaul operation has already proved its worth. Cases qualifying for it are:

a. The patient suffering from hernia nuclei pulposi whose back also shows signs of degeneration. These signs are, primarily, the flat-oval wide vertebral canal, the transitional vertebra, functional listhesis and anomalies of nerve tissue and surrounding membranes.

b. If the disc was of normal width before the herniatomy, the space between the vertebral bodies will in all probability become narrowed after discotomy and to our mind it is mistaken policy to wait and see whether, as a result, the foramen will become so narrow as to compress the root (Norlén, p. 17, Armstrong, pp. 45, 55 and 99, Cobey, pp. 43–45). We think that the necessary precautions should be taken during the herniatomy itself.

c. All cases in which, through posterior lipping, spondylarthrosis, etc., the spine shows a tendency to narrow the epidural space.

d. All cases of early osteoporosis, with the consequent danger of foramen narrowing.

e. All cases of paresis, atrophy, micturition disturbances (because these usually point to lesions of more than one root).



Roots L.4 and S.1, also the asymmetrically implanted L.5 roots, freed *bilaterally by overhaul. (Durography of a wide lumbar sac)*

to distinguish between 1) an overhaul operation as an extension of herniatomy and 2) an overhaul operation as an independent intervention.

Re 1. Opinions differ, as far as herniatomy is concerned, as to whether no more than is strictly necessary should be done, or whether there should be thorough exploration. The choice between these and the many intermediate stages between these extremes is governed by the relative success with which one is prepared to be satisfied if only the strictly necessary is done. As *we* see it, laminectomy is such an exceedingly important event to the patient that nothing should be left undone that could make the best possible job of it. Until very recently, we treated *all* herniatomy patients in this way and scored a very high percentage of good results. It is a higher percentage than that roughly given in the literature for herniatomy. When we compare these with the results of herniatomy during a period when we did not overhaul, we find that the results of herniatomy plus overhaul are about 6% better. The question now is whether herniatomy should or may be systematically supplemented by an overhaul operation as routine for the benefit of six out of every hundred patients.

By making it a rule to proceed with an overhaul operation after every herniatomy, one is undoubtedly doing too much in an unknown number of cases. We came to recognise by degrees the backs that decidedly required this operation, but it is as yet impossible to state the indication explicitly on the

at the site of the vascular system. Haemorrhages are rather profuse during osteotomy, partly because the bone is often osteoporotic. As has been stated, the joint is sometimes opened, but no definite harm was seen to result. If the vertebral canal is wider than usual, the pedicles of the arch are often thin. This must certainly be taken as a warning not to pursue the lateral exploration too far. Twice we had to remove a detached articular process. Once, post-operative radiographs showed that a transverse process had become detached. The person in question had had a trauma some years previously. It can scarcely be supposed that a healed fracture of the transverse process had fractured again; but, however that may be, the previously fractured process had again become loose after the lateral exploration. The patient suffered from rather severe disabilities in consequence, but they were entirely different from those for which the laminectomy was performed.

We consider an overhaul to be especially indicated when there are undoubted motor disturbances. Muscles being multiradicularly innervated, atrophy of the muscles and paresis strongly suggest lesions of more than one root. In such cases it is not enough to discover one hernia under one given root; a wary surgeon will also explore under the other roots, reasoning that one of the neighbouring roots must surely be functioning unsatisfactorily also. In point of fact, an abnormality is sometimes found at another level.

If a root compressed by a hernia is abnormally thick, or forms the common insertion of two roots, this would sufficiently account for the motor disturbance. All the same, it is safer to make sure that the neighbouring roots are free.

De Sèze (p. 1013) states that the L.5 root is impaired in 95% of cases of motor disturbances. The explorative method described affords ample exposure of the L.5 and S.1 roots. If it has been established that the S.1 root is quite free and it is assumed that motor disturbances point to something wrong with two roots, it must naturally be inferred that root L.4 is not functioning properly, if, that is, there are any motor disturbances. In such cases it was our practice to perform a foraminotomy for the L.4 root as well.

If motor disturbances are in doubt, the chronaxia test can be useful. With certain exceptions, prolongation of chronaxia points to a lesion of two motor roots and this, in our opinion, supplies the indication for an overhaul operation at several levels.

Rhizotomy

To relieve radicular pain, the aetiology of which is not revealed at exploration, one course that can be followed is section of the sensory fibres. For this the

f. All patients above the age of 45 who have to undergo a lumbar herniatomy.

An overhaul operation would *not* appear to be necessary in the following cases:

a. In association with the herniatomy of a normal spine if the patient is under 45 years of age.

b. If the disc involved is not unusually wide.

c. If a pre-operatively greatly narrowed disc does not bring about radicular symptoms.

d. In the absence of other indications for overhaul during re-operation for a one-sided syndrome initiated from the level of a disc which has collapsed after the first operation, there is a sound reason for leaving the contralateral side untouched; for, the absence of contralateral symptoms proves that the collapse of the disc on that side did not lead to compression of the root.

Guided by these principles, we have come to perform lumbar laminectomies upon patients whose symptoms formerly seemed to be untreatable because the diagnosis did not point to a hernia nuclei pulposi. We can understand why the correct indication was overlooked as far as the exploration for the then little-known nervous anomalies are concerned, seeing that experience in their surgical treatment has only recently been gained. It is less easy to understand why, after the failure of conservative treatment, middle-aged sufferers from spondylarthrosis should have been denied the chances of relief which surgical therapy proves to be able to provide in these cases. A root compressed by a bony spur is just as distressing as one compressed by a hernia; it is inconsistent to decompress it in the one case and not in the other.

An overhaul operation is a rewarding last resort for the middle-aged sufferer from spondylarthrosis whose health and vitality are unimpaired; the results amply make good for the inconvenience of undergoing this major operation. The indications for bilateral exploration and exposure of the structures adjacent to L.4, L.5, S.1 and S.2 are painful stiffness in the lower part of the spine, indeterminate radicular symptoms in both legs associated with varying reflex disturbances, often without sensory disturbances, wasting and atrophy of the thighs, nocturnal lumbar pain and micturition disturbances.

We have complemented herniatomy with an overhaul in a mitigated form approximately 1000 times Some of the "degenerative" backs thus explored were among the herniatomy-plus-overhaul group. The number of primary indications for exploration and overhaul of a degenerative back without hernia exceeded one hundred. It can be said with truth that in a follow-up of several years we did not find that any of the patients who had undergone this major operation were any the worse for it

It has its drawbacks, however. Lateral exploration involves manipulation

at the site of the vascular system. Haemorrhages are rather profuse during osteotomy, partly because the bone is often osteoporotic. As has been stated, the joint is sometimes opened, but no definite harm was seen to result. If the vertebral canal is wider than usual, the pedicles of the arch are often thin. This must certainly be taken as a warning not to pursue the lateral exploration too far. Twice we had to remove a detached articular process. Once, post-operative radiographs showed that a transverse process had become detached. The person in question had had a trauma some years previously. It can scarcely be supposed that a healed fracture of the transverse process had fractured again; but, however that may be, the previously fractured process had again become loose after the lateral exploration. The patient suffered from rather severe disabilities in consequence, but they were entirely different from those for which the laminectomy was performed.

We consider an overhaul to be especially indicated when there are undoubted motor disturbances. Muscles being multiradicularly innervated, atrophy of the muscles and paresis strongly suggest lesions of more than one root. In such cases it is not enough to discover one hernia under one given root; a wary surgeon will also explore under the other roots, reasoning that one of the neighbouring roots must surely be functioning unsatisfactorily also. In point of fact, an abnormality is sometimes found at another level.

If a root compressed by a hernia is abnormally thick, or forms the common insertion of two roots, this would sufficiently account for the motor disturbance. All the same, it is safer to make sure that the neighbouring roots are free.

De Sèze (p. 1013) states that the L.5 root is impaired in 95% of cases of motor disturbances. The explorative method described affords ample exposure of the L.5 and S.1 roots. If it has been established that the S.1 root is quite free and it is assumed that motor disturbances point to something wrong with two roots, it must naturally be inferred that root L.4 is not functioning properly, if, that is, there are any motor disturbances. In such cases it was our practice to perform a foraminotomy for the L.4 root as well.

If motor disturbances are in doubt, the chronaxia test can be useful. With certain exceptions, prolongation of chronaxia points to a lesion of two motor roots and this, in our opinion, supplies the indication for an overhaul operation at several levels.

Rhizotomy

To relieve radicular pain, the aetiology of which is not revealed at exploration, one course that can be followed is section of the sensory fibres. For this the

root(s) conducting the pain has (have) to be located. This can be done by monoradicular stimulation under a local anaesthetic, without intradural anaesthetization; the patient is not always able to give reliable information, either from nervousness or on account of variations in the neural innervation patterns.

Rhizotomy is indicated if the radicular pain is severe and the condition of the field of operation does not suggest that the explorative manipulations have already cleared up the radicular symptoms. Sensory (and motor) fibres are also sometimes severed when the condition of the root is expected to cause persistence or recurrence of radicular symptoms.

Rhizotomy is a rewarding expedient for undefinable radicular pain. The results are encouraging as far as the abolition of pain is concerned. It is usually possible to prevent motor neural loss and the patient soon becomes accustomed to sensory disturbances if these persist.

This operation should be selectively sensory; that is to say, the motor fibres should be spared. *Intradural* section has the advantage of permitting exploration of the contents of the dural sac, but there are serious objections to it, *viz.*,

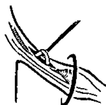
a. Spinal fluid fistulas are more likely to be formed if the dura is opened.

b. Outflowing spinal fluid and inflowing air cause the patient distress for several days.

c. Exposure to air seriously increases the risk of arachnitis.

d. The intricacy of the method lengthens the operation considerably.

e. It is more difficult, we think, to find the right root and to distinguish between sensory and motor fibres than by rhizotomy in the root sheath.



Rhizotomy in the root sheath

We systematically employ the last-mentioned method. With the help of the patient's reactions, the affected root is located by monoradicular mechanical stimulation. At about 5 mm from the dural sac the sheath is opened along 3 to 5 mm. The position of the sensory fibres is dorsal. They are lifted out of the opening with a thin, blunt hook and tested for sensory or motor function, fibre by fibre, mechanically or, if necessary, electrically. When the patient intimates pain but notices no shock in the extremity, that particular fibre is severed. It, or the bundle, is drawn a few millimetres out of the opening and is extirpated, mainly from a central direction and along approximately 5 mm. As a check, mild stimulation is applied to the root distal to the opening, when the patient should no longer experience pain, but there is still some muscular jerking.

Rhizotomy in the root sheath has much to recommend it, *viz.*,

a. There is no leakage; the opening is distal to the spinal fluid barrier.

b. The small opening does not require to be sutured

c. It is a simple, short procedure putting little strain either upon the surgeon or the patient.

d. There is no reason to fear intradural arachnitis.

e. Motor and sensory fibres are easily and reliably distinguishable.

f. There is no post-operative distress for the patient.

g. Even after intradural anaesthetization selective section is possible, though admittedly not quite so sure.

h. The results are not only as good as, but better than those produced by the intradural method (see p. 635).

i. Lesion of the arteria radicularis is impossible; hence the complications for which that is thought to be responsible are ruled out.

De Sèze and co-workers (p. 1788) have interesting things to say about the arteria radicularis. This vessel enters the dural sac through a separate opening, supplies mainly the motor fibres, gives off a small side-branch to the sensory bundles and in some cases supplies 1, 2 or 3 metameres of the spinal cord. Closure of the arteria by a hernia (as represented in their accompanying illustrations) near the insertion of the root sheath into the dural sac is said to be the cause of motor neural loss symptoms, burning sensations and variations in the clinical symptomatology. Ischaemia of the conus is also liable to occur and, more particularly, is thought to be responsible for the multisegmental character of some disturbances They say, rightly enough, that "ce mécanisme de l'ischémie nerveuse, radiculaire ou médullaire par compression vasculaire d'origine discale" has been repeatedly invoked to explain neural syndromes associated with *cervical* disc lesions.

Persona view.

We know from former experience in intradural lumbo-sacral rhizotomies that an arteria radicularis was sometimes [illegible]

but, as f[illegible]

occur sul[illegible]

plicate the post-operative course of every laminectomy Reischauer attributes such pre-operative or post-operative complaints to lesions of parasympathetic fibres in the L.5 root; we thought (*loc. cit.* p 142 (398)) that a disturbance of the collateral circulation in the lower extremities was induced by lesions of vessels in the epidural space.

Several points of De Seze's hypothesis need clarification, *viz*,

1. De Sèze draws the hernia half under the root sheath and half under the dural sac, *i e*, the place where he assumes that the arteria radicularis enters into the dura The vast majority of hernias are situated in the lateral recess, laterally How can hernias in this position seal off the arteria radicularis? If the hypoth[illegible] some of the cases [illegible] up the arteria in its c[illegible] arteria

Following De Sèze *et al.*

radicularis has already joined the root when it enters the dural sac. We ourselves have seen (during rhizotomy in the root sheath) the arteria radicularis entering the root sheath about 3 mm away from the dural sac. Even at this somewhat more laterally situated entrance it would be difficult for the lateral hernia to reach the arteria.

2. Only three of the four lumbo-sacral roots are accompanied in their intradural course by an artery of any size. (This is a personal observation which can be verified in almost any intradural exploration.) Hence the hypothesis is not valid for a hernia under the root without the arteria radicularis.

3. Microscopic abnormalities pointing to the consequences of vascular occlusion are not found in the nerve-root compressed by a hernia. It is inconceivable that severe symptoms should be produced by ischaemia of the nervous system without leaving demonstrable traces of the consequences of ischaemia. De Sèze does not mention these microscopic changes in the roots.

4. The presumed ischaemia of the conus through vascular occlusion in association with a lumbo-sacral hernia has not been demonstrated microscopically. However, ischaemic lesions after obliteration of part of the blood supply to the spinal cord (thoracic arteria radicularis) *are* known (see Woodard and Freeman, p. 63).

5. It may be asked whether the obliteration of part of the blood supply from a small arteria radicularis lumbalis would not be compensated by the superabundant collateral blood vessels of the spinal cord.

6. If the conus became considerably ischaemic, would not the clinical picture be richer in symptoms?

7 The most caudal root artery to supply the lumbar-sacral part of the spinal cord is the *arteria radicularis lumbalis magna*, so called, which on both sides accompanies one of the roots from thoracic 8 to lumbar 3 inclusive. The artery has never been found more caudally; the arterial blood supply to the spinal cord *via* arteries along roots below L.3 is insignificant (H. Felten, Dusseldorf, in an address at Freudenstadt, 1956, supplemented by personal communications to the author. Also see Lazorthes and co-workers, p. 113)

It may therefore be assumed that a hernia under roots L.4, L.5 and S.1 cannot exert more than trivial influence upon the arterial blood supply of the lumbo-sacral cord by obliteration of the arteria radicularis concerned. It also follows from this that such a contingency is not to be feared from a vascular lesion during rhizotomy of the L.4 and lower caudal roots.

Clearance of as many vessels as possible from the epidural space and interruption of the nervi sinuvertebrales in the foramen temporarily bring about partial denervation of painful joints. They certainly alleviate low back pain, but it returns in about six months' time, because the cut was distal to the ganglion and the nerve fibres therefore regenerate.

The indications for *chordotomy* have to be carefully considered. It is only a minor operation, performed through a small exposure of about 4 cm (to save the shoulder muscles), only the lower edge of one arch having to be nibbled away. Painful, constricting sensations in the arm can be prevented by cutting in between the insertions of the radicular filaments of roots Th.1 and Th.2.

We think unilateral chordotomy is preferable to sensory rhizotomy of a lumbar-sacral root if arachnitis is expected, or if a graft would have to be removed to make the nerve-root accessible. Spurling (1958, p. 104) is of the same opinion.

Fusion operation

Just as rhizotomy is a rewarding means of relieving undefinable radicular pain, so is a fusion for ill-defined low back pain. Controversy on this measure derives partly from the neurosurgeon's reluctance to hack away the graft, which he has to do in order to reach the firmly embedded hernia, and partly from the difficulty experienced by orthopaedic surgeons in fixing a bone graft when the spinous process and arch have been removed in a previous herniatomy.

Our personal experience with this method was gained largely from cases in which it was necessary to remove a hernia from underneath a graft. In view of very special indications, arthrodesis was only occasionally performed after a herniatomy. We were encouraged to impose this restriction upon ourselves by the results of a colleague (Mr. Hoeberrechts, Canada, Neurosurgeon formerly of Nijmegen) who, performing a combined herniatomy and arthrodesis in collaboration with the orthopaedic surgeon, found the graft to be a complicating hindrance and moreover, in a control series, saw no better results than of herniatomy without fusion. There are other colleagues, however, who are satisfied with the effect of the combined methods.

The divergence of opinions appears from the following brief excerpts from the literature

Barr (Lewin, p. 867) 50 combined method 68% good, 28% mild sciatic symptoms.

Bradford and Spurling (p. 115): "Spinal fusion is an adequate procedure when only the intervertebral discs are involved".

Burns and Young (p 245): Herniatomy + fusion, given special indications, produces good results in 50% of cases.

Cloward (p 166) His method of fusing the vertebral bodies is the treatment of choice (a high percentage of complete long-term cures (over 85%). He states that spinal fusion operations have a high percentage of failure (see Spiegel, 1955, p. 346)

Davis (p 424) Always performs herniatomy plus fusion.

Dibeley *et al* (Spiegel, 1957, p. 325) The results following disc surgery and fusion are 10% better than in disc removal alone.

Lewin (p 837): comes to no conclusion after surveying the literature. Armstrong's reserved attitude to fusion operations (p 213) is represented as a positive standpoint in favour of fusion (Lewin, p. 841)

Armstrong (p 213). "The results of the extirpation of the nucleus combined with immediate arthrodesis seem to be if anything a little worse than those which are produced by simple removal of the diseased nucleus". He refers to a considerable amount of discomfort and inconvenience in the long after-treatment following fusion (p. 212)

Davidoff (p. 610): One in 50 cases needs a fusion. "Poor judgment and worse surgery to subject 98 patients to unnecessary fusions for the sake of 2 of the 100. [illegible]".

[illegible] good mobility one year after fusion following lumbar disc excision is not necessary as a primary measure".

Muuro (see Spiegel, 1957, p. 327): considers fusion contraindicated as a part of hemilaminectomy done to relieve compression.

Milikan (see Spiegel, 1955, p. 344): ....herniatomy or herniatomy + fusion; no difference in results.

Scuderi and Khedroo (see Spiegel, 1956, p. 193): present an orthopaedic surgeon's point of view. They state that the hope that spinal fusion will cure backache with pain radiating to the leg is an illusion. It subjects the patient to an operation which carries a relatively high incidence of failure; it is painful and it prolongs convalescence.

Spurling (1958, p. 100): ...never herniatomy + fusion as a primary procedure. ...A congenital anomaly does not provide a special indication; a localised arthritis is no indication.

Unander (p. 127): 46 times fusion after herniatomy (22 times of which there was multiple disc degeneration; this is about 50%, whereas generally only 10–15% is reported). This is the summary of his cases:

After-treatment:	3 months plaster jacket;		
	2 months corset;		
	7 months no work.		
Results.	Good	65.2%	
	Improved	6 5%	
	Worse	28.3%	
Complications	Necrosis		2
	Fracture		10
	Pseudarthrosis		6
	Disc degeneration above the graft		4
		out of 46 patients	22

We considered it necessary to give this short review because of the importance of the question as to whether there should be fusion or no fusion. It teaches us the following:

1. The indication for fusion after herniatomy is not supported by the results.
2. Except perhaps for the fusion of the vertebral bodies as mentioned by Cloward, there is not a single method of fusion which deserves preference above all others.
3. Post-operative treatment is prolonged and troublesome.
4. Complications are by no means rare.

Our experience comes from a group of patients who had first undergone a

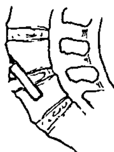
fusion operation elsewhere. The impression we received from these can be summed up thus:

1. In about 30% the graft breaks or goes septic and therefore, in approximately 12%, had to be removed again.
2. Of the remaining 70% about half benefited as far as the low lumbar pain was concerned, but few, if any, as regards radicular symptoms.
3. When the results are negative, the low back pain persists or symptoms recur after a few months, often quite different in character. There is then frequently found to be degeneration of another disc one level above (or below) the fused part of the lumbar spine; at functional examination there is then generally found to be a functional listhesis. This and the new disc degeneration act reciprocally, the tendency to displacement, however, being primary.
4. An autogenous bone graft seems preferable to one of banked bone. The patient often suffers prolonged discomfort from the wound made to obtain the piece of bone.

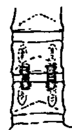
Little experience has been gained in bone grafting between the transverse processes. Some consider it to be a complicated procedure.

The findings at re-exploration of the roots after the introduction of bone chips are condemnatory out of hand.

Internal fusion between the vertebral bodies after discectomy by the retroperitoneal approach seemed promising at first, but, when these cases are followed up, the long-term results are found to be disappointing, even when the method has been applied for spondylolisthesis, for which it seems the chosen method (1).



1



2

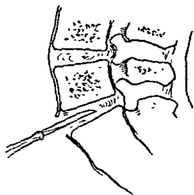
The same can be said of fusion of the vertebral bodies by the laminectomy approach (2).

The fusion involving destruction of the small vertebral joints produces the best results.

5. It would seem unreasonable to mobilise the patient at an early date after fusion; one cannot expect sufficient consolidation in two to three weeks. If the result is nevertheless satisfactory, this is in spite of the (unnecessary) fusion.
6. Instability of the spine as the cause of low back pain constitutes the most reasonable indication for fusion. Either pre-operatively or after a herniatomy we try to rectify the instability by all suitable means, *i.e.*, exercises, correcting faulty walking habits, normalising the position and movements of the spine, getting the patient to change his mode of life, to resume work, to take a

course of physiotherapy and maybe even massage. We think the physiological reconditioning of the muscular system, by which it is enabled to resume its supporting function, is more important than anything. If asthenia or the parlous post-operative condition of the sclerotic muscles makes these efforts abortive, a large orthopaedic jacket is still contraindicated because, taking over the function of the muscles as it does, it merely causes them to deteriorate. The proper compromise to our mind is a small canvas band, in an attempt to level out the incongruity between the task required of the supporting frame and that which it is still capable of fulfilling if it retains its mobility. If this likewise fails, then a fusion operation becomes imperative. Just because its mobility is then sacrificed for the relief of pain, it follows that, from the moment the indication for a fusion is admitted, the demands made on the frame have to be drastically restricted. To avoid other complications, notably a functional listhesis, the indication for a fusion operation at the same time implies that the patient is condemned to run in second gear for the rest of his life.

Herniatomy from the anterior (retroperitoneal) approach is a curious procedure with or without fusion, as will be apparent from the following facts. A correct level diagnosis is obtained by complete clinical examination in approximately 65% of cases. Even supposing that in the remaining 35% the hernia is found in the expected localisation by mere chance in half those cases (17½%), we must still allow for the repeatedly established fact that multiple hernias occur in about 12½%. This being so, the probability of finding the pain-provoking hernia upon exploration at one level is 65 + 17½ — 12½ = 70%.



This also holds for the anterior approach, through which, however, it is scarcely possible to explore for a second hernia. In an unknown number of cases the nucleus lies detached in the epidural canal, or there are other stenotic complications which cannot be seen in the ventral aspect *and certainly cannot be cleared away*. This is as good as saying that in more than 30% herniatomy from the ventral approach must inevitably fail; and this seems to be corroborated by the not entirely satisfactory follow-up records and the opinions expressed in the literature (Descuns and Garre). If herniatomy by the anterior approach were to fail in much less than 30% of cases, we should be forced to argue that, either the factual material collected down the years respecting hernia nuclei pulposi – on which this calculated percentage of 30 is based – is misleading,

or that surgical treatment is indicated in many cases too freely, since some of these 30% of patients appeared to recover with their hernia still *in situ*. It is our guess that the future will show up clearly the shortcomings of herniatomy by this approach.

The surgery of the degenerative back

By this we mean an attempt to restore the back to the best possible condition under the circumstances by surgical means.

1. This attempt will consist, in part, in removing a predisposing element to stenosis. We can extirpate prolapsed disc tissue, chisel off posterior lipping, widen the lateral recess, perform foraminotomy and canalotomy, etc. as described above.

2. We avail ourselves of the opportunity afforded by exploration and, if needed, herniatomy to get rid of as many pathogenic factors as possible. Even if the patient only presents the symptoms of a hernia nuclei pulposi at the moment, it would be short-sighted merely to remove the protruding nucleus, while leaving, say, a malformed arch or very swollen arthrotic joint untouched. The period at which these and other conditioned abnormalities manifest themselves and their gravity are determined endogenously; if they were left in place they might easily produce symptoms at a later date.

3. Besides these, there are constitutional anomalies which only begin to make themselves felt at maturity or in advanced age. In the event of the syndrome



Overhaul operation of a patient with a degenerative back, trimmed wide dural sac, decompression of root anomalies in the lateral recess, foraminotomy, canalotomy.

of a canal stenosis, the L.V, L.IV and L.III arches are removed, as recommended by Verbiest. Other examples are the fanwise extension of the pedicles of the arches, especially in the presence of transitional vertebrae, malformations of the intervertebral foramina, owing to which the roots may be compressed, a more than physiological arching of the posterior margin of the sacrum. The arch itself may be abnormal in shape. In a young woman the L.V arch was shaped like a buckled ring, yet the bone was not osteoporotic, nor did the joints show any arthrotic changes; and there was no evidence of a trauma. Owing to this deformity, the vertebral foramen was narrower than usual; with this predisposition to stenosis, the slightest protrusion of the disc would have been enough to cause root compression, which, in fact, did occur. Some arches are congenitally detached, as we saw in three cases; after extirpation of the loose part the patient was again able to bend backwards normally and straighten out after stooping. This is different from the traumatically fractured arch, kissing spines and periostalgia. The characteristic feature of these three abnormalities is the backache caused by every backward movement – hence including straightening from the stooping position – especially any movement involving twisting and hyperextension. Kissing spines are effectively remedied by nibbling off the spinous processes affected. Periostalgia of the arch or the spinous process, which often causes severe local pain upon pressure, is an abnormal condition to which the orthopaedic surgeon draws attention. It is treated simply and effectively by nibbling away the spinous process and scraping the periosteum from the appropriate arch. We know personally of two cases in which, at the orthopaedic surgeon's advice, mystifying low back pain, resistant to all other treatment, yielded to this.



4. The surgical treatment of anomalies of the contents of the vertebral canal depends upon the findings. It is a matter of experience that radiologically visible signs of degeneration, such as spina bifida occulta and an abnormally flat-oval wide vertebral canal, are quite often associated with anomalies of the cauda equina and adjacent membranes.

An *ectopic ganglion* is dissected out of its adhesions; a ganglion imbedded in bony tissue is chiselled out of adjacent structures. The same applies to *abnormally thick roots* and *roots with one common insertion into the dural sac*. We made it a rule that anomalies should debar primary rhizotomy. It was not only because decompression of the anomaly itself produced reasonably satisfactory results, but chiefly because we feared that rhizotomy might induce unforeseen gross

neural loss symptoms. The morphology and function of an anomalous nervous system may be so different from what one is accustomed to, that experience in the rhizotomy of normal roots would avail little in coping with the situation. At the worst, neural loss symptoms might ensue of comparable magnitude to the symptoms treated. In three cases of re-operation a rhizotomy was performed in the root sheath of a root anomaly and in two cases intradurally. Fortunately, the neural loss symptoms turned out to be purely and selectively sensory. In two cases, however, they were more extensive than it is legitimate to expect after the loss of the function of one sensory root.

As a rule, foraminotomy goes no further than widening the entrance to the foramen. Treatment of the anomalies just mentioned often necessitates nibbling the intervertebral canal until it is entirely open, in order to make enough room.

It is advisable to bear in mind that these anomalies are often associated with disc lesions.

The wide dural sac

When confronted with an abnormally wide, or pseudo-cystic lumbar dural sac, we were faced with the question as to whether this state of affairs was pathogenic. In cases of low back pain and radicular symptoms, the aetiology of which could not be convincingly proved, we assumed that the root system had been pushed by the pseudo-cystic dural sac into the lateral recess, as easily happens when the vertebral canal is wide. At first we merely widened the lateral recess but, when it was found that, after further lateral clearing, the roots were pushed by the wide dural sac further and further into the lateral recess, the obvious course was to try to disengage the roots from the lateral recess by moving them towards the middle. We eventually succeeded in doing this by reducing the size of the dural sac. The fact that the roots were now drawn from the recess and from the foramina in a medial direction seemed to argue in favour of our point of departure, *viz.*, that a large dural sac pushes the roots into the recess. The dimensions of the pseudo-cystic wide dural sac were reduced to about a third or half of its original size. This was done by taking in the cystic, distended dorsal part with 6 to 10 tangential sutures, care being taken not to enclose the roots. Even if, through faulty technique, the suture should pierce the dura, there is little danger of a cerebrospinal fistula, as the folds in the dural sac would cover the perforation. In patients displaying no other changes, the measure described above often had a favourable effect upon radicular symptoms and back pain. As we gained more experience, we came



to realise that, besides the mechanism mentioned, there is another one through which the pseudo-cystic lumbar sac may produce symptoms. It had already struck us that persons afflicted in this way experienced strange, jerky sensations at the base of the back when certain movements brought them forcefully on their heels. We attributed this to the local protrusion of one of these pseudo-cystic abnormalities through locally increased pressure. After removal of the ligamentum flavum with or without the arch in between, a wide lumbar sac may be expected to distend more still under increased pressure. The continuous feeling as of something pressing deep to the wound after laminectomies would



thus be explained in these cases. The treatment of the dura as just described at the same time provides the answer for this second mechanism; for, the posterior aspect of the pseudo-cystic distension is drawn together into firm tissue and for this precisely that thin part of the dural sac is used which previously became distended through offering little resistance.

We therefore resorted systematically to this measure in manifest cases of a





Fairly wide dural sac in a wide spinal canal. (Endo- and epidural ethiodan one year after myelography.) The contrast shows posterior distension of the *untrimmed* dural sac.

pseudo-cystic lumbar sac, also if some other abnormality, such as a lateral hernia, had sufficiently accounted for the pre-operative syndrome, hoping thereby to lessen the chances of the occurrence of fresh post-operative symptoms. If the dural sac was not exceedingly wide, we merely covered it with a strip of Spongostan *soaked in Globenicol*. As time goes on, this is transformed into a skin around which connective tissue grows, *forming* a firm partition between the dural sac and the muscles above; the dural sac is thus restricted in its tendency to distend. Subsequently this "artificial ligamentum flavum" proved to have further advantages: Any blood from the muscles left after closure remains stationary above this layer, while the Spongostan itself possibly seals off *small* perforations in the dura. We have not seen a true cerebrospinal fluid fistula since adopting this expedient and no wound complications occurred due to the Spongostan left behind. From that time on, it has become a routine measure with us to leave a covering layer of Spongostan on the dural sac after every *laminectomy*.

Complications

It is sometimes difficult to tell the difference between complications and a post-operative disorder causing "recurrent" symptoms.

Haematoma within 24 hours is a complication. If, after 24 hours, blood collects between the muscles or under the subcutis, we do not consider this to be a complication; it often is not discovered until a puncture is made as a pre-cautionary measure. If cerebrospinal fluid is mixed with it, that again is a complication and, through the *formation of a cyst*, may produce "recurrent" symptoms. The percentage of *cerebrospinal fluid fistulas* and other complications yet to be mentioned is negligible (roughly 0.5%). Since the local application of antibiotics has become common practice, *wound infection* has become virtually a thing of the past.

A *lesion of the large blood vessels* (*loc. cit.* p. 593) through perforation of the disc's anterior wall can be lethal; Harbison (see Spiegel, 1955, p. 347) found this in 44% of 30 cases he collected, but the frequency is not known. Krayenbühl (p. 58) reports in 998 operations two injuries to the vena cava inferior; further, five cerebrospinal fluid fistulas with three resultant cases of meningitis, one spondylitis, nine cases of thrombophlebitis with four infarcts of the lung. *Micturition disturbances* (14%) and *motility disturbances* are usually transitory. In 20% we found post-operative muscle wasting, including 16% in which this existed pre-operatively. The peroneal musculature continued paretic for longer than 5 months in 2%. We have already referred to infection of the surgically treated disc (Lenshoek, p. 57, Turnbull, p. 469, *loc. cit.* p. 164 (141)).

to realise that, besides the mechanism mentioned, there is another one through which the pseudo-cystic lumbar sac may produce symptoms. It had already struck us that persons afflicted in this way experienced strange, jerky sensations at the base of the back when certain movements brought them forcefully on their heels. We attributed this to the local protrusion of one of these pseudo-cystic abnormalities through locally increased pressure. After removal of the ligamentum flavum with or without the arch in between, a wide lumbar sac may be expected to distend more still under increased pressure. The continuous feeling as of something pressing deep to the wound after laminectomies would thus be explained in these cases. The treatment of the dura as just described at the same time provides the answer for this second mechanism; for, the posterior aspect of the pseudo-cystic distension is drawn together into firm tissue and for this precisely that thin part of the dural sac is used which previously became distended through offering little resistance.



We therefore resorted systematically to this measure in manifest cases of a





Fairly wide dural sac in a wide spinal canal (Endo- and epidural ethiodan one year after myelography.) The contrast shows posterior distension of the *untrimmed* dural sac.

Failure to extirpate a disc after injury to (coagulation!) the annulus fibrosus or posterior longitudinal ligament.
Failure to clear stenotic factors (vertebral margins, posterior lipping, narrowed foramina, bony spur formation in canal);
constricted arches; fanwise extension pedicles.
Intervertebral narrowing, with narrowing of the foramina as a result and no compensatory measures taken (foraminotomy, canalotomy).
Failure to widen the lateral recess in the presence of a wide vertebral canal.
Failure to recognise, decompress or otherwise treat anomalies of the root, ganglia and surrounding membranes.
Lesion of roots through traction, coagulation or incision (L.4!).
Silver clip left behind on root after intradural rhizotomy.
Intradural adhesions.
Extradural cysts – cerebrospinal fistulas.
Lesions of the arteries in the epidural space.
Damage to the joints.
Failure to excise a swollen arthrotic joint.
Fragments of bone left behind in the epidural space.
Fragments of ligamentum flavum left behind
Dense scarring.
Scar formation between skin, fascia and periosteum.
Instability of the spine
through sclerosis of much muscle tissue in unnecessarily large wounds;
through functional listhesis at another level.
Negligence in post-operative treatment, owing to which an intermediate between synostosis and pseudarthrosis persists at the site of the extirpated disc.
(References: Armstrong (p 222), Bradford and Spurling (p. 39),
Greenwood (p. 15), Lewin (p. 848), Muller (p. 352).
Sicard (p. 1323), Thurel (p. 26), Zukschwerdt (p. 168), etc.).

Post-operative Treatment = Prophylaxis to Avoid Recurrency

After an operation for an acute lesion, there still persists the morphological and psychic structure of the personality which, under the influence of precipitating factors, admitted or favoured the coming into existence of that lesion. Hence, even if the symptoms do not recur at the same place, the scene is set for a resurgence elsewhere in the body.

It is no idle precaution, therefore, to treat the total predisposing personality structure, if it can be done, and so to instruct the patient that he may learn to avoid the precipitating factors. These, as we have seen, consist mainly of strain and, sometimes, traumata. The patient must be taught, therefore, how to avoid strain. Spurling (p. 93) explains to the patient "that it is impossible for natural processes to restore the disc to its original state of integrity". The hernia can be removed, but it is up to the patient to do the rest. O'Connell (p. 8) considers it most important that the routine of exercises be continued indefinitely.

Recurrency is the most common "complication" of laminectomy. A distinction has to be made between recurrent hernia (fresh protrusion of nuclear tissue at the same level as that at which tissue has already been extirpated) and recurrent symptoms which could be produced from anywhere (*e.g.*, also by a new hernia at some other level). In about 3% of recurrency cases the patient himself intimates that the symptoms are *different* from what they were before.

In 1950 and 1951 there were 40 re-operations to 698 laminectomies. Of these, 19 persons (2.7%) underwent the first operation in the St. Ursula Clinic; it consisted of a herniatomy by the fairly conservative method still practised in the preceding years. At re-operation we found:

9 times lipiodol arachnitis;

once arachnitis without antecedent lipiodol;

7 times hernia at another level;

twice true recurrent hernia at the same place.

In a study of causes of failure, Greenwood (p. 20) comments on the importance of making sure that the roots are entirely untrammelled after a herniatomy, as became clear at re-operations. According to the literature, a percentage of 2 to 4% of necessary re-operations is quite usual: Brocher (p. 51) 4–6%; Spurling, 5.5%; O'Connell, 2.25%; Waris, 2%; Greenwood, about 9% (Spurling, p. 114). Opinions differ strongly on the necessity for re-exploration (*cf.* Armstrong pp. 209 and 222 and Zukschwerdt, p. 168).

Persistent or recurrent symptoms occurred immediately after the operation in 5%, after an interval free from pain lasting two weeks in 78%, after a similar interval longer than 2 weeks in 11%.

The causes of recurrent symptoms may be summarised as follows:

True recurrent hernia;
- (nucleus incompletely extirpated,
- remaining lateral parts of the disc,
- traces left behind in the oblique L V – S.I disc,
- undiscovered sequestra, *e g*, in foramen or canal;
- migrated sequestra under dural sac or to other levels;
- small hernias not felt by palpation,
- bud hernias overlooked during extradural (exploration).

Nuclear degeneration in a previously healthy disc.

Old hernia at another level
- faulty level diagnosis,
- erroneous determination of level of field of operation;
- multiple hernia,
- bilateral hernia;
  - (contralateral "recurrent" symptoms
    - in 7% without contralateral discotomy,
    - in 2% with contralateral discotomy)

added if the patient complains that the pain keeps him awake. This often helps to break down sleeplessness; one good night's rest seems to submerge the psychic disturbance evoked by the recent experience.

On the fourth day after the operation the patient may or must sit up.

On the fifth day he must stand and walk; exercises begin.

Exercises continued on the 6th day and for about one week in the hospital; thereafter for the rest of the patient's life!

Discharge between twelfth to fourteenth day; patient primed with advice as to mode of life.

Clinical exercises. During after-treatment of the surgically treated back, these are seldom combined with massage. Under the supervision of the physiotherapist the patient must perform precisely those movements which he finds most difficult to do. Painful stiffness must *just* be broken through, time and time again, but without forcing. Instead of being avoided, painful movements must be made deliberately. The more conscientiously this is done, the sooner will the pain diminish.

After discharge. We advise the patient to continue for a week to lead approximately the same life as during the last week in the hospital. After that, using his own commonsense, the patient may do everything as before in moderation, except lifting anything in a stooping position and diving head first. Hence there are no restrictions on walking, working or playing games, though it would not be sensible to start at once on vigorous sports and games (football, golf, ski-ing).

The advice we give is:

At once:	Indoor gymnastic exercises every day.
	Mild forms of sport, preferably swimming.
	Resume control of private business.
After 1 month:	Return to office.
	Return to light work which does not involve lifting objects while stooping.
	Take up household duties again, with certain restrictions.
After 2 months:	Resume heavy manual work, barring the lifting of heavy objects in stooping position.
After 6 months:	No restrictions.
	Continue physical exercises for all time.
	Keep going at sports and games.
	Change way of living (*not* job).
	If necessary, bring down weight, follow a diet, consume easily re-absorbed calcium in the form of buttermilk.

On the other hand, we have seen that lack of training, maladjustment, psychic and physical ageing processes, the neglect of personal physical culture during premature ageing, all adversely affect the condition of the back.

The obvious inference is that it is by training, exercises and sound advice that we should strive to restore the body to the best possible condition after the operation and *not* by a "go slow" policy. This is the line we pursued, and it was vindicated by the results.

The circumstances, of course, vary from individual to individual; there are some cases in which we are compelled by the individual disorder to depart from our general principles, examples being a herniatomy at L.II – L.III, premature osteoporosis, age, general weakness of the supporting frame, marked anomalies of movement and posture. In each individual case it is necessary to consider the capabilities of the patient and, if it must be, he and his medical adviser will have to put up with second-best.

At one end of the scale there are advocates in the literature of extremely cautious post-operative rehabilitation, *i.e.*, a plaster bed for months on end, plaster jacket, exhortations never to stoop down, never to do heavy manual labour, to change over to another job. Next come those who do rehabilitate, but with admonishments as to extreme cautiousness in mode of living. At the other end of the scale there appears to be a general tendency to keep the patient in bed for a short period only after the operation and to discharge him after about a fortnight. Early resumption of work, under mitigating circumstances at first, is advised, with a view to boosting up the psyche, especially in so-called "compensation cases", the prognosis of which is generally expected to be much worsened by neurotic reactions.

We agree with the most progressive advice on after-treatment, which is carried out as follows.

If the patient were to wish it, he would be allowed to walk back to his room or ward! (So far, this has never happened.)

Minimise the gravity of the operation by immediately granting some small concessions, such as coffee and smoking.

From the first hour, patient is to move his legs.

Patient to be allowed to move slightly, unaided, but not to turn over in bed.

After lying supine for the first four hours on the wound, he may lie as he wishes, even on his back.

Analgesics, including opium derivatives, not to be stinted during the first three days.

In cases of serious insomnia after the fourth day, we withhold all narcotics for one night, but give a larger dose the following night, with opium derivatives

added if the patient complains that the pain keeps him awake. This often hel[p] to break down sleeplessness; one good night's rest seems to submerge th[e] psychic disturbance evoked by the recent experience.

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Exercises continued on the 6th day and for about one week in the hospita[l] thereafter for the rest of the patient's life!

Discharge between twelfth to fourteenth day; patient primed with advi[ce] as to mode of life.

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	Continue physical exercises for all time.
	Keep going at sports and games.
	Change way of living (*not* job).
	If necessary, bring down weight, follow a diet, consum[e] easily re-absorbed calcium in the form of buttermilk.

According to reliable statistics, the number of laminectomies for a population of twelve million (the Netherlands) is approximately 1000 a year. If these persons were placed in a special category as far as their work and mode of life are concerned, it would increase every year by roughly 1000 persons. In one generation (about 30 years), the labour market would be embarrassed by an army of about 30,000 people. Let alone that this might be an insupportable social burden for the population to bear, under present circumstances, even, it is difficult for the individual to find light work. It is fortunate that, as Brocher says (p. 52), the safest thing for the surgically treated sufferer from low back pain to do is to return to his former occupation, because he is trained in his own job and can therefore avoid strain more skilfully than a novice.

Does it not strike the reader as strange that satisfactory results should be obtained by two such contrary methods of after-treatment, one prolonged, tentative and wary, the other active early on? This antithesis could be formulated differently: After discectomy, Spurling opens the superior and inferior surface of the adjacent vertebral bodies; it is our practice to chip off the vertebral margins and the cartilaginous laminae; that is two different descriptions of virtually the same thing. It is our opinion that this measure after discectomy favours pseudarthrosis, an opinion that rests on reports by Foltz (p. 469) and what we have observed ourselves after operations. In Spurling's opinion (expressed personally to the author), the measure promotes the formation of a synostosis between two vertebral bodies. We would adduce Jenkner's experiments on animals (p. 451) in support of that view, despite the fact that real synostosis is not seen in man.

We suggest the following explanation: As with all callus formation, rest favours synostosis and movement induces pseudarthrosis. Hence our earlier statement that good results are obtained with early mobilisation after arthrodesis of any kind, in spite of the fusion; *i.e.*, through the failure of the synostosis. If one wishes to fuse a certain part of the spine, one must give synostosis a chance, so to speak, by prolonged immobilisation.

There are no statistics in the literature providing evidence of *better* results from immobilising, careful, prolonged after-treatment. Indeed, the general tendency towards adopting the more active line points rather to the contrary.

Our primary object is to make the patient stronger; secondarily we try to do this by combating the symptoms. The object of our method is to improve the patient's condition by decompressing the nervous system and by promoting the normal mobility of the spine. That is why we are all for early, active rehabilitation. Even if, medically, both methods were to produce results of equal value, we think the active one is preferable because of its social advantages.

Post-operatively, prophylaxis begins in order to hold off a fresh lesion. The maintenance of health and strength after the operation is over, depends on

the doctor managing the patient during after-treatment;

the personality of the patient and the way he carries out the doctor's orders;

the patient's constitution and the condition of his back.

In a word, where back disorders are concerned, the doctor, whether it be general practitioner or specialist, bears the responsibility for the *total* personality of the patient.

Results of Surgical Treatment

The ideas set down in this study rest on the experience gained in more than ten thousand clinical and polyclinical cases (case histories of the St. Ursula Clinic at Wassenaar, St. Canisius Hospital and University of Nijmegen).

We do not know what the ratio of back disorders to surgically treated hernia is; according to some authors it is something between 2% and 5%. In our opinion the ratio of clinically probable hernia to hernia resistant to conservative treatment, needing surgical treatment ranges from 20% to 25% (see p. 598).

The author's own surgical experience covers upwards of 2000 cases (St. Ursula Clinic, Wassenaar, St. Canisius Hospital and University of Nijmegen, Red Cross Hospital, Djakarta, 1948). In 6% there was no follow up.

Some patients with degenerative backs were asked to report on their feeling of well-being by answering a questionnaire (files in St. Canisius, Nijmegen), as many of these exceptional cases were spread all over the country and could not, therefore, easily be re-examined personally.

Herniatomy

Some favourable data from the literature

	Per cent.			
	++	+	±	—
Armstrong, p. 221 (review of the literature)		62	17	
Barr		90		
Bradford and Spurling		74		
Brussatis	42	34	9	
Love, 1st series		80		
2nd series		90.4		
Burns and Young		80	11	
Key		85		
Lenhard		83		
Petit Dutaillis and De Sèze		86		
Spurling, 1st series		79		
2nd series	60.4	22.1	12.8	
De Vet (p 502)		82	8	

No real difference through changing herniatomy technique

		+
Dandy:	Loose fragments removed	76.9
(Lenhard, p. 427).	Curettage and discectomy	71.1
	Multiple discectomy	64.0
Waris (p. 106).	Same opinion.	

	Herniatomy in selected hernia cases	*Laminectomy without hernia*
	+	+
Senning and Sjoquist	96.8	67
Waris	94	70
Lindgren	95	

THE AUTHOR'S RECORDS OF HERNIATOMY

The following figures are intended merely to afford an impression and have no greater value than that. Our study of the degenerative back brought out once again the diversity of back disorders, which makes it difficult, if not impossible, to draw up significant statistics, nor do we believe that data of that kind have much statistical value.

General information

			Per cent.
			+
	2000	laminectomies for low back disorders	70–82
of which	500	herniatomies for low back without overhaul	78
and	1000	herniatomies for low back with overhaul	84
		(Many degenerative backs with psychic disturbances; several methods of surgical treatment.)	

Selected groups

		Per cent. ++	+	±	—
200 hernias in "normal" backs					
Conservative herniatomy (follow-up 2–3 years)	Subj.	52	82	30	18
	Obj	60	85	25	15
Fitness for pre-operative occuption and way of life after 2–3 months			54	40	6

200 hernias in "normal" backs

		Per cent. ++	+	±	—
Herniatomy + overhaul operation; active training after fourth post-operative day (follow-up 2–3 years)	Subj.	65		23	12
		88			
	Obj.	77		14	9
		91			
Fitness for pre-operative occupation and way of life after 2–3 months			77	21	2

500 hernias (without differentiation of constitution or method employed, but without special overhaul for the elderly)

Age	*Cases*	+	±	—	+ %	± %	— %
< 25	46	38	6	2	83	13	4
25—35	176	142	12	22	82	7	11
> 35	278	167	87	24	62	32	6
	500	347	105	48			

Duration of symptoms	*Cases*	+	±	—	+ %	± %	— %
6 months to 1 year	122	97	13	12	81	10	9
1 to 2 years	102	83	13	6	83	12	5
2 to 5 years	166	107	46	13	65	28	7
5 to 10 years	43	26	4	13	60	9	31
Longer than 10 years	67	31	29	7	46	43	11
	500	344	105	51			

Results classified under occupation and sex

		Cases	+	±	—	+ %	± %	— %
Sedentary work	♀	20	13	5	2	65	25	10
	♂	63	60	2	1	95	3	2
		83	73	7	3			
Long hours of standing	♀	49	32	4	13	65	9	26
	♂	124	85	27	12	68	22	10
		173	117	31	25			

		Cases	+	±	—	+ %	± %	— %
Housework	♀	161	75	29	57	47	18	35
Arduous manual work	♂	83	52	25	6	63	30	7
		244	127	54	63			
Women		230	120	38	72	52	17	31
Men		270	197	54	19	73	20	7
		500	317	92	91	63.4	18.4	18.2
The prognosis of patients in military service is favourable (young, mental stability, selection for constitution and condition, active post-operative training):								
Men in the Forces		28	24	3	1	90	7	3
Same favourable prognoses (see Warts, p. 101). By contrast, the prognosis for the nursing staff proved to be far less promising:								
Nurses		16	9	3	4	56	19	25
Classification of the result of operations on *psychically disturbed patients*:								
Roughly		40				40	30	30
Prognosis of *compensation cases* Spurling, p. 109. "incredibly bad"								
Aitken and Bradford.						65		
		Cases	+	±	—	+ %	± %	— %

The dissimilarity between the various methods employed for the first operation and the many guises in which back disorders are manifested make it impossible to collect the results of *re-laminectomies* in a statistical form that is truly representative.

We studied the particulars of 117 re-operations. Ostensibly they constituted approximately 3% to 5% of the whole of our material, but several patients who were advised to undergo another operation did not do so, or were operated upon elsewhere, while 42 of these 117 cases had had their first operation elsewhere. It may be said in general terms that a true recurrent hernia at the site of the previous operation is rare. The prognosis of the herniatomy of a fresh hernia at a different level is scarcely

worse than that of a primary one. The re-laminectomy for recurrency in the case of a patient primarily operated upon by another surgeon had a far better prognosis, understandably enough, since application of the same surgery as failed the first time is less likely to be successful than re-operation by another (better) method.

After the adoption of contralateral discectomy as a routine method, the number of contralateral recurrency complaints dropped to one third!

On an average, re-operations took place between two and three years after the first one. After 1949 herniatomy plus overhaul operation was adopted as a routine method. It was realised that the benefit of this procedure could only become apparent many years later by the diminishing indication for re-laminectomy; and in fact 19 re-operations had to be undertaken in 1950 + 1951, whereas in 1956 + 1957 only 13 cases (3 with overhaul, 10 without) of approximately the same number of laminectomies had to undergo surgery for the second time.

Results of laminectomies without herniatomy (negative exploration for hernia)

Authors(s)	*Incidence of negative exploration* %	*"Pure" disc herniatomy* + %	*"No disc herniation".* + %
Senning and Sjoquist	15	96.8	67
Waris (p. 92)	12	94	70
Shinning and Hamby	17		
Malmrose	15		
Friberg	12		
Zukschwerdt (p. 166)	(33.5)		Yet the majority of patients had no symptoms.
Krayenbuhl (p. 56)	2.4		

Personal investigations

Results of NEGATIVE EXPLORATION *for presumed hernia* (not including special indication for overhaul operation) according to follow-up after at least two years.

Cases	*Total result* + %	± %	— %
132 (about 5% of the whole series)	40.1	22.7	37.1

These were split up as follows:

Before 1949. No overhaul Cases	+ %	± %	— %	*After 1949: Overhaul not planned pre-operatively* Cases	+ %	± %	— %
42	21.4	26 2	52.2	90	48.8	21.1	30

Classification according to details in diagnosis

No overhaul Cases	+	±	—		Overhaul Cases	+	±	—
21	7	5	9	Typical case history for hernia	44	25	7	12
21	2	6	13	Explorative	46	19	13	14
42					90			
16	2	6	8	Myelography —	31	13	6	12
26				Myelography false +	59			
42					90			
3	0	0	3	Clinical				
				syndrome III – IV	3	1	0	2
21	5	5	11	IV – V	40	23	8	9
8	3	0	5	V – S.I	20	8	5	7
10				Not clear	27			
42					90			

Classification according to some aetiological details

Cases	+	±	—		Cases	+	±	—
29	8	7	14	Ligamentum flavum normal	55	23	13	19
11	2	4	5	Ligament hyperplastic or extensive in wide canal	20	14	4	2
9	2	3	4	Post-traumatic	9	3	4	2
5	0	1	4	Evident psychic disturbance	11	3	1	7
				Congested epidural veins:				
3	2	1	–	2 months after operation	4	3	1	–
	?	1	1	3–4 ditto		?	1	2
	–	1	2	6–8 ditto		–	1	3
				Spondylarthrosis	3	2	1	0
				Transitional vertebra	3	2	1	0
				Spondylolisthesis	6	3	1	2
				Canal stenosis	4	2	0	2
				Anomaly of cauda or surrounding membranes	18	10	2	6
				Spondylarthrosis (overhaul planned pre-operatively)	23	14	2	7

Classification according to some technical details in overhaul

Cases	+	±	—		Cases	+	±	—
1	0	1	0	Discotomy	11	8	2	1
				Infiltration of concealed disc with alcohol of 96%	5	1	0	4

					Cases	+	±	—
2	1	–	1	Decompression of the arch	1	1	–	–
2	1	1	0	Foraminotomy	46	30	12	4
				Foraminotomy + rhizotomy	20	11	8	1
				Foraminotomy + discotomy	3	3	–	–
				Foraminotomy + discotomy + rhizotomy	1	–	–	1
				Discotomy + rhizotomy	1	–	–	1
				Foraminotomy + rhizotomy + duroraphy	4	3	1	–
				Foraminotomy + duroraphy	3	2	1	–
				Duroraphy	8	5	1	2

RHIZOTOMY

Author(s)	*Cases*	*Fit for work* +	±	*Not fit* —
Norlén (p. 79)	15	10 Without pain	4 With some pain	1 some pain
De Vet (p. 2169)	120	75	21	18 (and 6 recurrencies later)

(General result from several indications)

Personal investigations (including many of De Vet's cases)

Prior to 1949 no overhaul — After 1949 rhizotomy + overhaul

Sensory rhizotomy L.5

28 cases *Result* +	±	—		*Intradural*			14 cases *Result* +	±	—
7	10	11	7	Sensory disturbances:	instep big toe	4	5	5	4
			9		lat. instep little toe	5			
			8		lat. lower part leg	4			
			2		thigh–hip	2			
			9	Motor disturbances:		4			
			8	Reflexes – or <:	Ach. t reflex	4			
			2		knee t. reflex	–			
			3		Ach. and knee	3			

4 cases +	±	—		*Extradural* in root sheath			41 cases +	±	—
2	–	2	1	Sensory disturbances.	instep big toe	9	30	9	2
			1		lat instep little toe	11			
			2		lat lower part leg	5			
			–		thigh–hip	–			
			2	Motor disturbances		5			
			1	Reflexes – or <	Ach. t reflex	15			
					knee	2			
					Ach. and knee	3			

Classification according to details in diagnosis

No overhaul Cases	+	±	—		Overhaul Cases	+	±	—
21	7	5	9	Typical case history for hernia	44	25	7	12
21	2	6	13	Explorative	46	19	13	14
42					90			
16	2	6	8	Myelography —	31	13	6	12
26				Myelography false +	59			
42					90			
3	0	0	3	Clinical syndrome III – IV	3	1	0	2
21	5	5	11	IV – V	40	23	8	9
8	3	0	5	V – S.I	20	8	5	7
10				Not clear	27			
42					90			

Classification according to some aetiological details

Cases	+	±	—		Cases	+	±	—
29	8	7	14	Ligamentum flavum normal	55	23	13	19
11	2	4	5	Ligament hyperplastic or extensive in wide canal	20	14	4	2
9	2	3	4	Post-traumatic	9	3	4	2
5	0	1	4	Evident psychic disturbance	11	3	1	7
				Congested epidural veins:				
3	2	1	–	2 months after operation	4	3	1	–
	?	1	1	3–4 ditto		?	1	2
	–	1	2	6–8 ditto		–	1	3
				Spondylarthrosis	3	2	1	0
				Transitional vertebra	3	2	1	0
				Spondylolisthesis	6	3	1	2
				Canal stenosis	4	2	0	2
				Anomaly of cauda or surrounding membranes	18	10	2	6
				Spondylarthrosis (overhaul planned pre-operatively)	23	14	2	7

Classification according to some technical details in overhaul

Cases	+	±	—		Cases	+	±	—
1	0	1	0	Discotomy	11	8	2	1
				Infiltration of concealed disc with alcohol of 96%	5	1	0	4

Sensory rhizotomy L.5 + S.1

Intradural, 3 cases: + | ± 1 | − 2

Intradural, 1 case: + | ± | − 1

Extradural, 4 cases

1	Sensory disturbances:	Instep big toe	1
3		Lat. instep big toe	4
3		Lat. lower part leg	4
2	Motor disturbances:		–
	Reflexes:	ankle and knee <	1
		ankle — or <	3
		knee <	1

Sensory rhizotomy L.4 + L.5 + S.1

Intradural: + – | ± 1 | − –

Sensory disturbances: Lat. lower part leg
and instep big toe
ankle reflex

Sensory rhizotomy S.1 + S.2

Intradural: + – | ± 1 | − –

No neural loss symptoms
Sensory disturbances: Lat. lower part leg
and instep big toe

Sensory rhizotomy L.5 on both sides

Extradural: + 1 | ± – | − –

No sensory nor motor disturbance
Reflexes ankle and knee L < R

Sensory rhizotomy S.1 on both sides

Extradural, 1 case: + | ± ? | −

Intradural, 3 cases: + 1 | ± 1 | − 1

–	Sensory disturbances.	lat. instep little toe	2
		lat. lower part leg	2
		thigh–hip	1
–	Motor disturbances		—
	Reflexes:		
1		ankle – or <	2
		ankle and knee — or <	1

Sensory and motor rhizotomy L.5

Intradural, 2 cases: + | ± 1 | − 1

Extradural, 6 cases: + 1 | ± 2 | − 3

–	Sensory disturbances:	instep big toe	2
		lat. lower part leg	2
		lat instep little toe	1
		thigh–hip	1

Sensory rhizotomy S.1

11 cases +	±	−		*Intradural*			15 cases +	±	−
1	2	8	–	Sensory disturbances:	instep big toe	–	4	4	7
			7		lat. instep little toe	10			
			3		lat. lower leg	7			
			1		thigh–hip	1			
			1	Motor disturbances:		4			
			6	Reflexes – or <:	Ach. t. refl.	11			
			–		knee	1			
			2		Ach. and knee	1			

11 cases +	±	−		*Extradural* in root sheath			35 cases +	±	−
4	4	3	2	Sensory disturbances:	instep big toe	4	19	12	4
			5		lat. instep little toe	21			
			3		lat. lower leg	7			
			1		thigh–hip	2			
			1	Motor disturbances		4			
			8	Reflexes – or <:	Ach. t.	25			
			1		knee	3			
			2		Ach. and knee	4			

Sensory rhizotomy L.4

			Intradural 3 cases +	±	−
Sensory disturbances:	instep big toe	1			
Motor disturbances:	(not the same case)	1	1	1	1
Reflexes:	ankle —	1			
	knee —	1			
	<	2			

			Extradural 2 cases +	±	−
	ankle —	1	2	–	–
	knee				

Sensory rhizotomy L.4 + L.5

			Intradural 2 cases +	±	−
Sensory disturbances,	Instep big toe	1	–	1	1
	Lat. instep little toe	2			
	Lat. lower part leg	2			
Motor disturbances		1			
Reflex disturbances		–			

Results in some degenerative backs *

			Patient's intimation			
			Discharge from clinic	Follow-up verbal	Follow-up in writing	
W.	N 13966	d.b., wide vc., wide ds., narrow for., ect. gangl.	+			
W.	N 13364	d b., blood in r., thick r., wide vc., spina bifida	+			
H.	N 14792	st. dysr., (psych. disturbances) mop, thick rr., ect. gangl.	+			
.K.	N 9958	myel. and ro. unclear, cyst (melanoma) S.1 (hist. W4140)	+			
A.	N 14913	hernia, atypical nerve root areas, asym. root insertions	+			
.B.	N 12165	Varicosities, rht.	± to —			
Z.	N 7273	lysis ?, myel. –, wide ds., mop, detached arch	+			
K.	N 14635	wide ls.	+	+		
V.	N 12318	wide ls, r. thickening, ect. gangl.	+		+	XV
D	N 13101	atypical myel., relative stenosis vc., "pantiles" over wide vc., rr. vertically beside ds., alternating insertions, etc. gangl. (rht.) (psych. disturbances)	+		+	XIV
S.	N 12699	wide vc., wide ds., myel. –, hernia + "pantiles"	+		+	XIII
D.	N 12034	d.b (spina bifida), wide vc, wide ds., tr.v., myel. –	+		+ (±)	XII
A.	N 8492	d.b (spina bif.) wide vc., wide ds., mop, ect. gangl. myel ±, hernia	—		±	X

N	= Nijmegen	tr.v.	= transitional vertebra
vc.	= vertebral canal	adef.	= arthrosis deformans
for.	= foramina	st dysr.	= status dysrhaphicus
rht	= rhizotomy	ect. gangl.	= ectopic ganglion
asym.	= asymmetrical	myel	= myelography
ls.	= lumbar sac	ro.	= X-ray
ds	= dural sac	r. = root	rr. = roots
d.b.	= degenerative back	I–XLVII	= patient's letter number

Psych. st. means that patient was operated upon *despite* clear psychic disturbances

9

1 Motor disturbances:
Reflexes

ankle — or < 3
knee — or < 4
atrophy 1
anaesthesia dolorosa 1
3

Intradural
2 cases

Sensory and motor rhizotomy S.1

Extradural
2 cases

+	±	—
1	–	1

1 Sensory disturbances: lat. lower part of leg 1
lat instep little toe 1

1 Motor disturbances:
Reflexes: –

1
–

ankle and knee < 1
anaesthesia dolorosa 1

+	±	—
–	–	2

			Patient's intimation			
			Discharge from clinic	verbal	Follow-up in writing	
J	N 12459	(psych. st.) wide vc., pantiles, wide ds., bud hernia in foramen	+	+		
v.G	N 12784	d.b., tr.v., sp. bif., asym. narrow vc., myel. –, hernia +	+	+	+	XXXa
W	N 12260	deformed atroph.r., myel. ± hernia L.IV–L.V, myel. –, bud hernia L.V–S.I	±		—	XXXI
v.O.	N 12869	(psych.st.), asym vc., bilateral ect. gangl., hernia	+		+	XXXIII
R.	N 12157	d.b., asym. wide vc., pantiles, constricted arch, ect. gangl. in lateral recess, rht.	+		?	
M.	N 13085	d.b., wide vc., wide ds., myel. –, bud hernia	+		+	XXXIV
B.	N 13820	d b , wide vc., wide ds., invading cleft in arch	+		+	XXXV
D.	N 12798	wide vc., pantiles, wide ds , rht.	±	+		
B.	N 10700	(psych.st.) sp.bif., asym.vc., wide ds , r. anomaly (thick and abnormal course)	±		+	XXXVI
V.	N 13769	pantiles, wide ds. invading cleft in arch, r. anomalies: one stem L 5 and S.1 empty root sheath !	+		+	XXXVII
V.	N 11020	wide vc., wide ds., ect. gangl. myel. – + Emigration			+	XXXVIII
v d W.	N 14013	d.b., wide vc., sp. bif , wide ds.	±		±	XXXIX
v H.	N 13256	d.b., sp. bif., adef., myel. –, hernia +, ect gangl , thick r., asym. insertion	+		+	XL
B	N 15277	pantiles, wide vc., malformation of r	+		+	XLI
v.d C	N 15003	d b., sp bif , wide vc , detached arch, wide ds , thick r.	+		±	XLII
d W.	N 12513	d b., wide vc , pantiles, wide ds.	+		∓	XLIV
V.	N 12257	d b , wide vc., wide ds., myel. –, hernia +	—		—	Xl

			Patient's intimation		
			Discharge from clinic	Follow-up verbal	in writing
P.	N 12781	d.b., tr.v., flat oval vc., "pantiles", myel. –, prolapse ±	+ (fusion)	±	VIII
A.	N 12299	d.b., flat oval vc., pantiles, r. thickening, myel. – hernia + !	+	+	VII
V	N 12844	(psych disturb.), thick discoloured rr., ect. gangl., atypical nerve root areas, myel. +, hernia +	+	+	VI
C.	N 12776	wide, myel –, bud hernia	+	+	IV
G.	N 12709	d b, wide vc, wide ds., myel. –, hernia +, sanguinolence	+	±	III
R.	N 12710	d.b., wide vc., wide ds., pantiles, myel +, hernia, rht.	+	—	I
H.	N 12567	d.b., arthrosis deformans, pantiles, thick rr., ect. gangl. (psych. disturbances)	+	±	XVI
B.	N 14661	d.b., adef., varicosity	±	+	XIX
J.	N 14535	d.b, tr. v., wide vc., wide ds.	+	+	XVII
B.	N 13855	d.b., tr.v, atypical nerve root areas, hernia L.V–L.VI and L.VI–S.I	+	+	XXI
B.	N 12982	myel +, hernia, ect. gangl. (= gangl. neuroma hist. W. 3340), rht, fusion	±	+	XXII
E.	N 14694	sinus pilonoidalis, epidermoid cysts	+	+	XXIII
K.	N 12357	d.b, tr.v., spina bif, wide vc, pantiles, wide ds, thick rr., asym insertion, r L.5 lacking	+	+	XXIV
R.	N 14334	d b.,asym insertion,duplication r L 5 right, hernia	+	+	XXVI
B.	N 13856	d b., sp bifida, wide vc, pantiles, wide ds, myel. –, hernia	+	+	XXVII
G.	N 12166	thick rr, asym. insertions, abnormal course, duplication S.1 ?	+	+	XXVIII

SUMMARY AND CONCLUSION

Part I. Points of Enquiry

Formerly, the problems presented by low back pain and radicular symptoms (manifestations of a malady which, for convenience, we shall call back disorders or a weak back) were approached from too one-sided a standpoint, for there was a time when surgeons bunched the several variants of this malady under the generic names of "lumbago", "coup de fouet", "Hecksenschuss" and the like. After orthopaedic surgery had developed from general medicine into an independent specialism, back disorders were often regarded by its exponents as arthrosis deformans of the lumbar spine, as some manifestation of rheumatism, or were associated with some clearly congenital deformity. Neurologists at first considered this syndrome to be an expression of sciatica or sciatic neuritis.

Once the clinical picture of hernia nuclei pulposi had been inaugurated, all specialisms having to do with the malady in question were apt to docket such back disorders as a manifestation of hernia nuclei pulposi. The study of back disorders was hampered by the lack of a pathognomonic symptomatology. Owing to the uncertainty of diagnosis, the data on symptomatology and treatment of the hernia not verified at operation were and still are insufficient and, therefore, unsatisfactory. Many riddles remained to be answered, even in cases of operatively confirmed hernia. The persistence of symptoms after the excision of a hernia and their cessation, occasionally, after a so-called "negative exploration" pointed to other conditional factors involved in back disorders.

Without having to turn to records in the literature or special investigations, it is apparent, merely from a few clinical particulars, that the state of irritation assumed to prevail in back disorders affects the two-thirds distal part of the root where this runs in the root sheath (between the dural sac and the ganglion). It also seemed reasonable to assume that a multiconditional genesis must be responsible for the postulated state of irritation. This implied that the pathogenic condition of spinal disorders should be sought in the lateral recess of the spinal canal. This realisation induced us to give our special attention to the lateral recess and this eventually led to a modification of the surgical technique hitherto commonly adopted. As a result, many factors predisposing to limitation

			Patient's intimation: Discharge from clinic	Follow-up: verbal	Follow-up: in writing
E.	N 13847	myel. ±, hernia –, r. anomalies: one stem L.5 and S.1, sensory and motor fibres in separate root sheaths	+	+	XLV
V.	N 12977	pantiles, ganglion anomaly in lateral recess	+	+	?
R.	N 13953	d.b., tr. v., sp. bif., wide vc., pantiles, wide ds., nerve root cyst	+	+	XLVI
M.	N 2774	d.b., wide vc., roots L.5 and S 1 from one insertion	+	±	XLVIII
't W.	N 12240	flat wide vc., pantiles, myel. + +, hernia ±	+	+	II
S.	N 12411	(psych. disturb.) wide vc., wide ds., multiple abortive meningoceles (dura rents), ventral insertions of nerve roots	+	±	XLVII

	Discharge +	Discharge ±	Discharge —		Follow-up +	Follow-up ±	Follow-up —
52	+	±	—	43	+	±	—
	43	7	2		30	10	3
	82.7%	13.4%	3.9%		69.7%	23 3%	6.9% (9 unknown)

Bud hernia nuclei pulposi in patients with a degenerative spine

		Myelography	Discharge from clinic	Follow-up: verbal	Follow-up: in writing
R.	N 7468	+ III – IV	+		
E.	N 12716	—	+	+	IX
v.K.	N 6171	—	+	+	V
C.	N 12776	—	—	+	IV
D.	N 14002	?, arthrosis deformans	+	±	XVIII
d.L.	N 13637	—, prot. 1.48 $^0/_{00}$, (psych. st)	+	+	XX
W.	N 13551	—, arthrosis deformans	+	+	XXIX

the) *epidural space* are discussed in relation to the occurrence of spinal disorders.

Stenosis of the spinal canal produces a specific syndrome; its discovery and description (Verbiest) acquire significance within the framework of this study, despite its rarity, as it shows that a developmental disturbance may predispose the subject to a specific syndrome in adulthood.

We dwelt upon the constitutionally determined *flat-oval shape of a wide spinal canal* which, unlike stenosis of the canal, occurs in very many members of the human race. We pointed out that hitherto no mention has been made in the literature of this particular shape of the human spinal canal as a developmental disturbance, nor to its significance as a predisposing factor to backache. Paradoxically, the width of this canal, an impression of which can be obtained from an antero-posterior radiograph, gives an idea of the lack of space in the lateral recess which may well predispose to root compression. Without being causally pathogenic, this configuration paves the way for attack upon the spine by adventitious changes, such as lipping, small disc prolapses and the like, which, of themselves, would not have passed the pathogenic threshold. The correlation of this particular shape of the vertebral column with other anomalies, its demonstrability by X-ray and its clinical significance are dealt with fully in the chapter on Radiology (Part III).

The use and meaning of the word "degenerative" are discussed. The developmental disturbances referred to may be taken as manifestations of a degenerative constitution. It must never be forgotten, however, that numerous abnormalities identical with the endogenous ones may equally well be exogenous. Furthermore, developmental disturbances may also occur as the result of an endogenous weakness *and* an exogenous derangement. In this study "degenerative" is used first and foremost to describe all disturbances in development resulting from an abnormal quality of certain genes in the pattern of chromosomes. Next, we denote as "degenerative" all derangements which ensue from developmental disturbances in the first phase of life through the action of exogenous factors, these being morphologically indistinguishable from the analogous genetic abnormalities. Hence the use of the postulate "degenerative" is defensible *ex definitione*. We found, moreover, that the disorders considered to be typical of a status degenerativus, according to the literature, were in many cases hereditary and are also denoted as degenerative in medical parlance. These disorders usually point to a development in a descending line; certain aspects of them, however, suggest refinement of structure, so that in some cases "degenerative" is synonymous with "cultivated", or even "over-cultivated". This is even more so when decadent cultural influences have been implicated in the pathogenesis.

of space were observed, including anomalies of the cauda equina and surrounding membranes. Not only did these explorations clarify our views on the pathogenesis of back disorders, but the modification of our technique proved to have favourable therapeutic effects.

Several facts, different in kind, seemed to suggest that a certain type of constitution predisposed its bearers to back disorders. The association between gross constitutional deformities and the predisposition to spinal trouble had already been pointed out in the past; but these anomalies are too rare to account for all the unsolved problems. It was fairly evident that the postulated constitution would be revealed in more or less common, unobtrusive manifestations which used to be considered "normal", or else would manifest itself in aspects of the human being which do not show morphologically in surgical explorations (psyche, function, motility).

Part II. Literature: Survey and Discussion

The vast, almost unsurveyable literature was searched as diligently as possible for views expressed as to a constitutional predisposition to spinal disorders, some aspects of which may be represented as follows:

Asthenia – one of the endogenously predisposing factors referred to – is characterised by an inferior diathesis of muscles and ligaments and is often accompanied by neurocirculatory syndromes and psychic behaviour typical of asthenia.

The *anatomy of the spinal column* is discussed, with special reference to its function as part of the supporting frame. Long before the clinical picture of hernia nuclei pulposi had become known, congenital deformities of the vertebrae were regarded as a predisposing factor to, or even as the cause of, low back pain.

Decalcification of the skeleton may give rise to low back pain. Under certain circumstances this pain is part of the involution syndrome, the period and severity of whose manifestation are determined, in part at least, endogenously. Prophylaxis against and treatment for involutional osteoporosis require primarily hormonal substitution rather than administration of absorbable calcium.

In the author's view, formed after study of the relevant literature, primary pathogenic significance is not to be attached to hypertrophy or hyperplasia of the *ligamentum flavum*.

The *posterior longitudinal ligament*, the *nervi sinuvertebrales* and the (contents of

spinal complaints and disturbed psycho-motoricity in so far as this is brought about by factors other than organic-cerebral and/or organic-spinal abnormalities. In the author's opinion, further investigations might throw more light on this matter; he also believes that this relationship has hitherto been overlooked because in the past the question, as such, was not raised.

Part III. The Author's own Material and Conclusions

The symptomatology of backache is dealt with *in extenso*, attention being paid mainly to the symptoms of an endogenously weak back and also to indeterminate syndromes induced by arthrosis deformans and the like; in each case, however, the known symptomatology of the clinical picture presented by hernia nuclei pulposi is invoked for comparison. In this part of the work, disturbances in movement and posture, neurological peculiarities, psychopathological behaviour and radiographic changes are brought forward consecutively in so far as they can be related to the symptomatology of back disorders.

It transpires that there are no special *mobility and postural disturbances* of the back that could be qualified as pathognomonic symptoms of this or that spinal disorder. But, if the back stiffens on forward flexion of the spine, a scoliosis exists (or follows upon movements) and a corkscrew phenomenon occurs, this provides strong evidence of a disc lesion. Many suggestions have been brought forward to account for the blocking of a back. It is not inconceivable that a back may become blocked by the "imprisonment" of the bobbin-shaped thickening of root plus ganglion behind a prominence in the lateral recess of the spinal canal when the patient stoops.

Neurological examination does not disclose pathognomonic symptoms which would be serviceable for the differential diagnosis of spinal disorders. Neither the neurological picture (dealt with here in the sequence of sensibility, motoricity, reflexes), nor the anamnesis on the irradiation of pain provides reliable indications from which the level of a lesion of the root or disc could be inferred; although the combination of some signs suggests a likely localisation, it does not provide a watertight prediction either of the level or the nature of the lesion. The more pronounced are the morphological signs of endogenous weakness in the spine, the less valuable is the sensory examination for locating a lesion of the root.

Variations and anomalies of the sensory innervation patterns were discovered by mechanical monoradicular stimulation in patients given a local anaesthetic; they were found frequently in association with manifestations (elsewhere in the body as well) of an unstable constitution. Our findings led

Another distinction emphasized is between "degeneration as a biological process" and "the degeneration of tissue", both being denoted as degeneration, since in most European languages there is no good alternative. In the literature, however, a distinction is certainly made between degeneration of the disc, in the sense of an endogenous predisposition to degeneration (or disturbed trophism, or inferior diathesis of an intervertebral disc), and degeneration of the disc, in the sense of a regressive change of tissue. We think it would be clearer to state that the two processes take place side by side in the disc; that is to say, inferior diathesis predisposes to a regressive change of the tissue.

The writer then comes to the conclusion that a hernia nuclei pulposi is brought about by degeneration of the disc at a period of life in which the tissue of the disc is still soft and gelatinous. The degeneration of an older, sclerotic disc, on the other hand, will proceed as a "merciful" process, seldom causing a hernia; then backache will be produced by lipping, narrowing of the foramina and other changes around the collapsed disc.

The *anomalies* in the roots, ganglia and adjacent membranes, found in great numbers during exploration of the lateral recess, are described in detail, mention being made of the predisposing part they play in the production of complaints and symptoms.

A separate chapter is devoted to the concepts *constitution, condition, predisposition* and *precipitation*. It is perhaps apt to point out, with reference to the chapter on traumatology, that constitution is never the cause of an illness; it is merely the predisposing conditional factor. If we wish to picture the degenerative back as a "constitutional disease", it is clear from the foregoing that exogenous pathogenic influences (illness or trauma) are essential to the actualisation of the constitutional disposition.

After studying the literature, we find that the majority of the authors accept a constitutional predisposition to spinal disorders and that there is agreement on the influence of strain and stress upon their precipitation. Further, it is so evident that most authors consider a disorganised motoricity as the result of defects in the (lower) extremities to have a pathogenic effect upon the whole supporting apparatus of the spine, that the present writer has refrained from reporting similar cases from his own experience exemplifying the connection.

Assuming that motoricity disorganised in this way adversely affects the back, it is reasonable to suppose, we think, that motoricity disturbed in some other way will do the same. There are known examples, but there does not appear to be any reference in the literature to a significant correlation between spinal disorders and disturbed motoricity as the result of neurological ailments (pareses and ataxia). Nor does the literature mention the relation between

varying function of the nervous system is rooted in the quality of the human constitution; some phenomena then follow an unusual course and often cannot be adequately explained. This unfamiliarity and inexplicability far too often tempt the physician to brand the symptoms as psychogenic. The word "functional" is correctly used to describe variations in function; but it is also used, incorrectly, to qualify the reactions of the patient, in many cases wrongly assumed to be inadequate, with the tacit implication of a psychically functional (*i.e.*, psychogenic) disturbance, which in fact it is not.

The *psychic* pictures to be observed in many people with the morphological signs of an endogenously weak back are fully described (constitutional nervousness, neurotic and psychopathic behaviour, consequences of a deranged temperament). The fact was brought to light by a scrutiny of clinical histories that the diagnosis "psychically disturbed" was made post-operatively about five times as often as pre-operatively. It may be that too little attention was paid to the psyche of back sufferers in the past; probably post-operative residual symptoms are branded too readily as psychogenic. Another point to be considered is whether the disappointment of finding complaints persist triggered off a latent psychic disturbance. The dynamisms by which psychical disturbances may be converted into spinal disorders receive full attention.

In regard to *diagnostic procedures*, we consider lumbar puncture for cerebrospinal fluid analysis to be a necessary part of the pre-operative routine examination. *X-ray examination*, on the other hand, should be resorted to only with the utmost discretion. A severely reserved attitude is advised in the matter of the indication for myelography, a method of examination which is conspicuous for its unreliability. Owing to the danger of ionising rays, this examination should be limited to a few exceptional indications.

Certain radiological facts are discussed, from the interpretation of which the diagnosis and treatment of back disorders can benefit. Special attention is paid to the dynamisms responsible for a so-called "functional retroposition" of vertebrae, which must be differentiated from true spondylolisthesis and pseudo-spondylolisthesis. As a rule, the disc in between proves to be primarily intact. Functional listhesis is the cause of innumerable cases of low back pain.

The chapter on symptomatology concludes with answers to a number of questions, including those on the dynamisms which are involved in the production of low back pain and radicular symptoms; also on the predisposition of the lower section of the lumbar spine to these disabilities. It is explained elsewhere that microscopic examination of the roots of the cauda equina does not reveal changes suggestive of an active process of inflammation that could account for these signs and symptoms.

to the hypothesis that every root of the cauda equina is multisegmental in composition. This, if correct, implies that the areas of the skin determined by classical physiology in various ways on the assumption that the root is monosegmental are, therefore, not dermatomes but root areas.

It is to be inferred from the same findings and considerations that the human lumbo-sacral roots contain fibres which function like fibres of neighbouring roots (transitional fibres). In persons with transitional vertebrae and other signs of degeneration, these transitional fibres occur in greater numbers, so much so that the sensory innervation patterns can be said to exhibit anomalies rather than variations. This theory was tested by several known physiological experiments, including the dermatome determination and antidromal conduction of stimuli, when it appeared that the theory regarding transitional fibres is not incompatible with the experimental results. The applicability of the theory to the motor fibres was also investigated. On the basis of our ideas respecting transitional fibres, we approach the explanation offered by classical physiology for various phenomena (*overlap, dissociated sensory disturbances* through lesion of a posterior root) from a fresh angle.

In view of the frequent coincidence of variations and anomalies in the neural innervation patterns with morphological anomalies of the nervous system and of the supporting frame of the spine, it is suggested that this may possibly not be a mere chance concomitance. If so, the very presence of anomalies in the neural innervation patterns might be taken as a sign of degeneration.

Arguments are advanced in support of the statement that transitional fibres in neighbouring roots are offshoots of those cells which found their way into adjacent ganglia during the division of the neural crest. These transitional fibres also have a part to play in the re-innervation of a denervated end-organ; the recovery of function in a peripheral area behind an incurable (or not yet healed) root lesion can be explained in part by the residual function of transitional fibres in the neighbouring roots which are still intact.

The variability of the neurological picture, which is so common in the endogenously unstable human being, called for a discussion of other influences which are apt to obscure the syndromes. The difference is therefore considered between surface and depth sensibility, as also some problems raised by referred pain. With the same object in mind, we dwelt on differences in the experiencing of pain from individual to individual; for, the value of the sensation of pain as a neurological symptom (within the framework of the symptomatology of the degenerative back) declines when the sense of pain has already been disturbed by other factors which are not implicit in the morphological component of a spinal disorder. Evidence is in fact adduced to show that a

the possible results of, and the objections to, chiropractice, neural therapy and other special methods of conservative treatment are considered.

Lastly, several paragraphs are devoted to certain aspects of laminectomy. The statement that fewer and fewer hernias are removed by surgery is contested on the basis of particulars published by the Central Bureau of Statistics in Utrecht, The Netherlands, which record an annual increase in such operations and therefore furnish proof to the contrary. The course of events which accounts for these contradictory contentions is the following: At first, when the clinical picture of hernia nuclei pulposi became known, a larger percentage of a smaller number of sufferers from spinal disorders was admitted for surgical treatment on the basis of a doubtful or erroneous indication. Subsequently, a more strictly established indication reduced this percentage considerably; this notwithstand ing, the total number of herniatomies increased because, when the possibilities inherent in surgical treatment became better known, a far larger number of patients consented to undergo the treatment.

Assuming the current technique of operation to be known, certain details of herniatomy and, in particular, of the overhaul operation are described. After studying the literature, the author comes to the conclusion that the results of a primary fusion operation combined with herniatomy are no better than those of herniatomy alone, for which reason fusion operations should, in his opinion, be performed secondarily in specially indicated cases only.

It appears to be difficult to decide whether herniatomy is indicated in the case of a patient who has been suffering for a short time from a severe hernia syndrome. It is to be inferred from the results that herniatomy is applied to best therapeutic effect upon young people for the removal of a fresh hernia. Accordingly, it is the author's practice to advise herniatomy for young people innocent of psychical disturbances and the morphological signs of degeneration if, after two weeks' absolute rest followed by trial mobilisation, the syndrome has not cleared up to a considerable extent.

The results of the surgical treatment of spinal disorders are tabulated according to details of aetiology and surgical technique, among other particulars. The experience of surgical treatment applied to patients exhibiting special aspects of a degenerative back is presented by condensed case histories in tabular form. It transpires that the surgical treatment of an uncomplicated hernia offers the best prognosis; it is also good, though not as favourable as for a classical hernia, in certain cases of arthrosis deformans, disc lesions with lipping, stenosis of the intervertebral foramen, etc., and also for the degenerative back in the strict sense, without a hernia.

In the chapter on *pathogenesis*, emphasis is laid on the fact that what is called a weak back is often a combination of several disorders. It is postulated as a hypothesis that the constitutional predisposition to affections of the spine might be traced back to a heritable disposition *via* culturally decadent influences.

In more than 2000 operatively verified cases, certain proof could not be produced in a single instance that an *injury* was responsible for a hernia nuclei pulposi in a previously healthy disc. The possible traumatic aetiology of spinal disorders is discussed in the light of this finding and the opinions expressed in the literature, combined with the view expounded in the preceding chapter. The conviction that there is an interplay between a weak-back constitution (and/or acquired conditions leading to it) and the injurious effects of a trauma pervades this whole discussion. Given a weak back, a relatively minor injury may produce effects which, in the case of an optimum constitution, could only have been brought about by a severe injury.

The opinions and conceptions laid down in the chapters on symptomatology and pathogenesis are gathered up and placed within the focus of an encompassing vision. It is pointed out that very many sufferers from low back pain have an almost normal spine, except for a few subclinical manifestations of an unstable constitution. Apart from some rare cases in which a trauma proves to be necessarily pathogenic, the origin of the ailment is obscure. Where there is a manifest unstable constitution, however, the connection between psychopathological behaviour and a disturbed psycho-motoricity is obvious. The reason suggested for this connection is that a certain labile personality structure involves repressed emotional stresses. These are externalised in abnormal muscular tension from which distorted postures and movements spring. The tense muscles of the back disturb the physiology of the spine and make it hypersensitive to the effects of minor injuries (accident prone), while the abnormal tensions in the spine may bring about "degenerative changes". The over-all derangement embraces, moreover, a) tendency towards abnormal metabolism, b) abnormal psychic behaviour, c) abnormal postures and movements, d) abnormal emotional stresses which are incarnated in the musculature and thus bring about unco-ordinated movements and postural anomalies.

The concluding chapter deals with *therapy*. The various kinds of conservative treatment and their results are discussed. In the author's opinion it is to some purpose to prescribe two weeks' complete bed rest for the treatment of a hernia syndrome, after which mobilisation should be tried out so that the doctor may decide what treatment is to follow

Although epidural Procaine infiltrations are of little therapeutic value, they can be recommended under certain specified circumstances. The indication for

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